The Journal of Thoracic Surgery

VOL. 18 DECEMBER, 1949 NO. 6

Original Communications

CHRONIC ATELECTASIS AND PNEUMONITIS OF THE MIDDLE LOBE

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ONE of us (D.L.P.), in 1945, became interested in chronic atelectasis and pneumonitis of the middle lobe of the right lung. Several patients were seen who had an obstruction of the bronchus of the middle lobe secondary to extrinsic pressure by calcified lymph nodes and resulting in atelectasis and pneumonitis of the middle lobe. Subsequently other patients were operated upon who had the same condition with the exception that instead of calcified lymph nodes the bronchus of the middle lobe was surrounded by large inflamed lymph nodes. In the last three years a total of thirty-two patients having chronic atelectasis and pneumonitis of the right middle lobe for periods varying from five months to twenty years has been seen. Twenty-nine of these thirty-two patients have been operated upon and a right middle lobectomy performed. A review of the symptomatology and the findings by roentgenography, bronchoscopy, and bronchography reveals a characteristic diagnostic pattern on the basis of which the diagnosis can be made preoperatively with a high degree of accuracy. In our opinion the frequency and characteristic pattern of this type of pulmonary suppurative disease justifies its separation as a clinical entity and stimulated our making this report.

PATHOLOGY

The essential pathologic feature of this condition consists of bronchial obstruction of the middle lobe bronchus with resulting atelectasis, pneumonitis, and fibrosis of the middle lobe. The bronchial wall has been found to be involved with inflammation and edema in most cases (Fig. 1). Enlarged peribronchial lymph nodes about the right middle lobe bronchus have been found in fifteen patients. These inflamed lymph nodes are large indurated nodes which in some instances completely surround the bronchus of the middle lobe at its origin from the intermediary bronchus. It is conceivable that these nodes which are probably secondary to inflammation within the right middle lobe produce pressure on the bronchus, thus increasing the obstruction of an already

Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

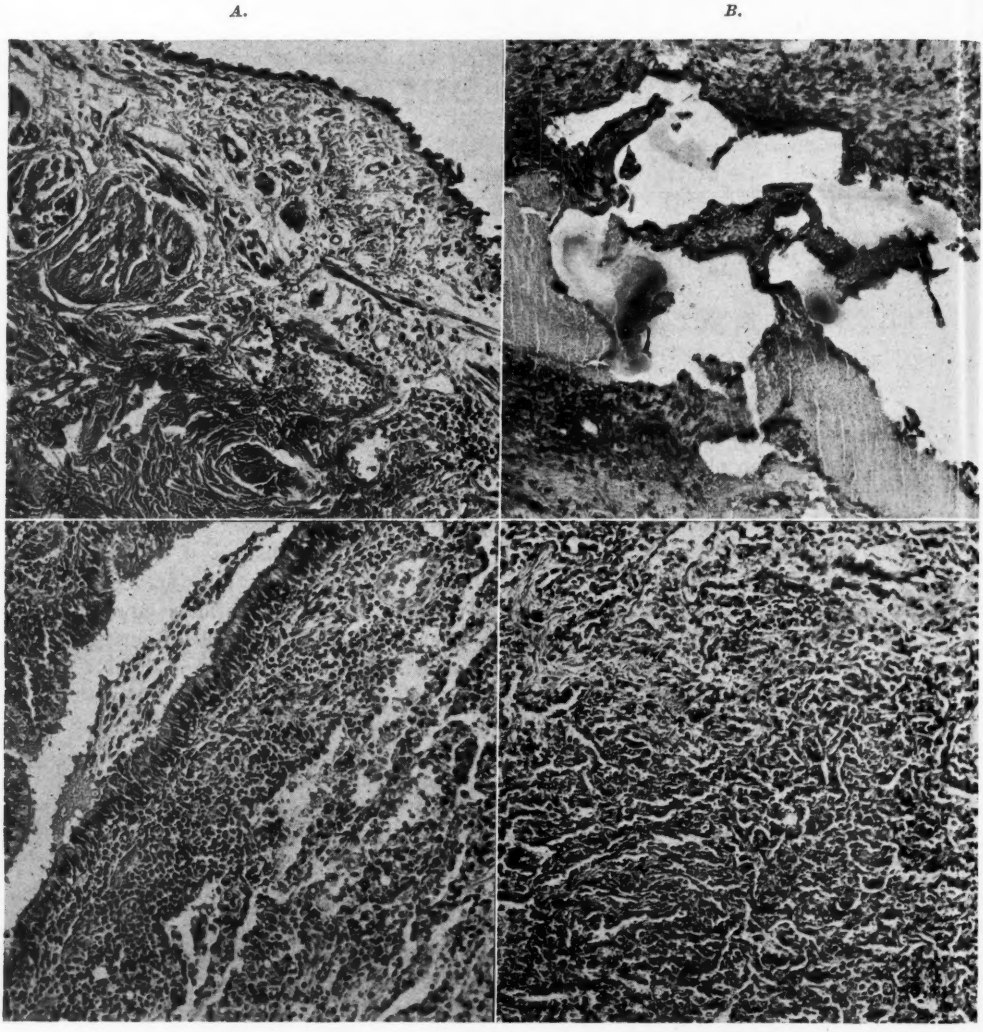


Fig. 1.—Photomicrographs (×100) of sections from the middle lobe of various patients having chronic atelectasis and pneumonitis of this lobe, illustrating: *A*, edema and inflammation in the wall of a large bronchus; *B*, a bronchus at the site of erosion by a calcified mass. Masses of hyalinized and calcified material are embedded in dense fibrous tissue which has replaced the mucosa of this bronchus. Chronic inflammatory cells of the lymphocytic type can be seen scattered throughout the connective tissue. Ulceration of the bronchial mucosa is evident, and in the vicinity of the bronchus, marked pulmonary fibrosis and chronic pneumonitis are noted. *C*, Section from lung tissue beyond the site of bronchial obstruction. Marked bronchiolitis is depicted with fibrous thickening of the bronchiolar wall and marked inflammatory infiltration. The adjoining lung tissue is atelectatic, and there is a severe chronic pneumonitis with fibrous thickening of the alveolar walls and infiltration by chronic inflammatory cells (lymphocytes, plasma cells, and macrophages). *D*, chronic atelectasis and pneumonitis.

inflamed and edematous bronchus. In a smaller group of patients (six) there were no enlarged lymph nodes in the hilum of the middle lobe, the bronchial obstruction apparently having been produced by edema and inflammation of the bronchial wall only. In eight patients, large calcified lymph nodes were found around the middle lobe bronchus. In several of these patients there was a complete ring of calcified nodes surrounding the bronchus. There is no question that these calcified lymph nodes were producing extrinsic pressure on the bronchus thus inducing obstruction. In four patients these stony lymph nodes had eroded partially through the bronchial wall (Fig. 1). This process, if allowed to continue, would undoubtedly result in a broncholith. Secondary to the bronchial obstruction complete or partial atelectasis occurs together with chronic infection. A chronic pneumonitis and fibrosis supervenes. Bronchiectasis eventually develops distal to the bronchial obstruction. Bronchiectasis, however, is not a prominent feature of the pathology but is only secondary to long-standing bronchial obstruction. It is considered entirely probable that inflammation and edema of the bronchial wall undoubtedly contribute to bronchial obstruction of the middle lobe bronchus, thus inducing atelectasis in the absence of extrinsic pressure by enlarged peribronchial lymph nodes or calcified lymph nodes. In two patients the bronchial obstruction was due to a foreign body reaction in the bronchial wall.

SYMPTOMATOLOGY

The cardinal symptoms of chronic atelectasis and pneumonitis of the middle lobe consist of chronic cough, pain, hemoptysis, dyspnea, and wheeze. Chronic cough was present in all of the thirty-two patients. In seven patients the cough was worse in dorsal recumbency, that is the patients complained of the fact that on lying down at night they developed severe coughing which was relieved by sitting in an upright position. Twenty-one of the thirty-two patients had pain which was usually located in the anterior portion of the chest, in many instances near the midline. Intermittent hemoptysis occurred in fourteen of the thirty-two patients. Hemoptysis varied from frank blood spitting to blood streaking of the sputum. Dyspnea was present in eight of the thirty-two patients, and a definite wheeze was described by the patient in six instances. Previous pneumonias constituted a prominent part of the clinical history. Twenty patients had had a previous pneumonia, nineteen of whom had had a pneumonia diagnosed two or more times. Fifteen patients complained of a constant low-grade fever. Eleven patients complained of easy fatigability. Frequent colds and loss of weight were present, respectively, in seven patients. Table I illustrates the incidence of these symptoms.

TABLE I. SYMPTOMS AND FEATURES OF THE CLINICAL HISTORY OF THIRTY-TWO PATIENTS

SYMPTOMS	NO.	HISTORY OF	NO.
Cough	32	Pneumonia	20
In dorsal recumbency	7		
Pain	21	Low-grade fever	13
Hemoptysis	14	Easy fatigability	11
Dyspnea	8	Frequent colds	7
Wheeze	6	Loss of weight	7

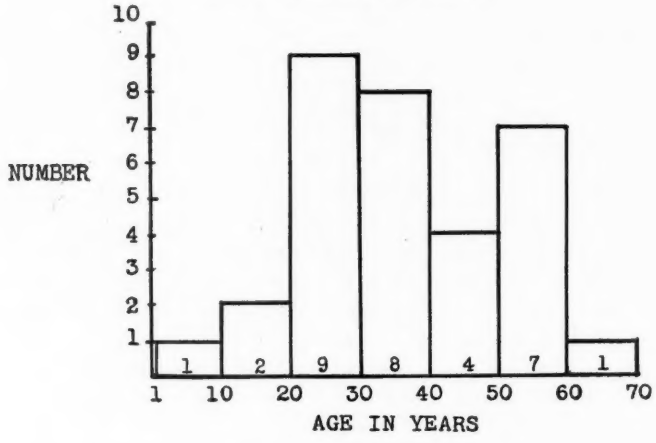
The duration of symptoms is illustrated in Table II and varied from five months to twenty years. It is to be noted that twenty-six patients had had symptoms for one or more years and that seventeen patients had symptoms for three or more years.

TABLE II. DURATION OF SYMPTOMS

UNDER 1 YR.	1 TO 2 YR.	3 TO 5 YR.	5 TO 12 YR.	20 YR.
6	9	10	6	1

The incidence as to sex revealed an almost equal division between males and females, 17 being males and 15 females. The age of these patients varied from 7 years to 62 years. Table III illustrates the age grouping.

TABLE III. AGE INCIDENCE



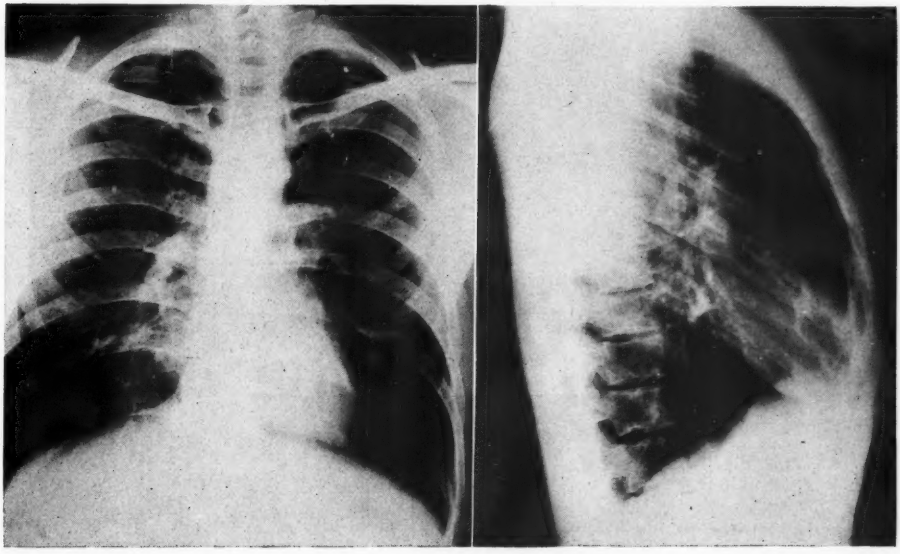
OBJECTIVE FINDINGS

The bronchoscopic findings consist of an edema, hyperemia, or induration of the mucosa of the bronchus to the middle lobe. Purulent secretion was seen arising from the middle lobe orifice, and in many patients the opening was markedly narrowed and slit-like in appearance. Some patients obtained temporary relief from the symptoms following a bronchoscopy performed for diagnosis. The bronchoscopic findings are not diagnostic alone but are of considerable value when taken into consideration with a careful history, roentgenographic and bronchographic findings.

TABLE IV. BRONCHOGRAPHIC FINDINGS

	NO.
Complete obstruction of bronchus	12
Incomplete filling of one segment	7
Close grouping of bronchi	4
Bronchiectasis	5
Total	28

The roentgenographic and bronchographic findings are very important in the diagnosis of chronic atelectasis and pneumonitis of the middle lobe. It is necessary to make right lateral roentgenograms in addition to the conventional posteroanterior and anteroposterior exposures. In many instances the atelectasis will not be visible on the conventional posteroanterior exposure only to be very obvious on the right lateral exposure (Fig. 2). An atelectasis of the middle lobe was diagnosed by the roentgenologist in nineteen of thirty-two



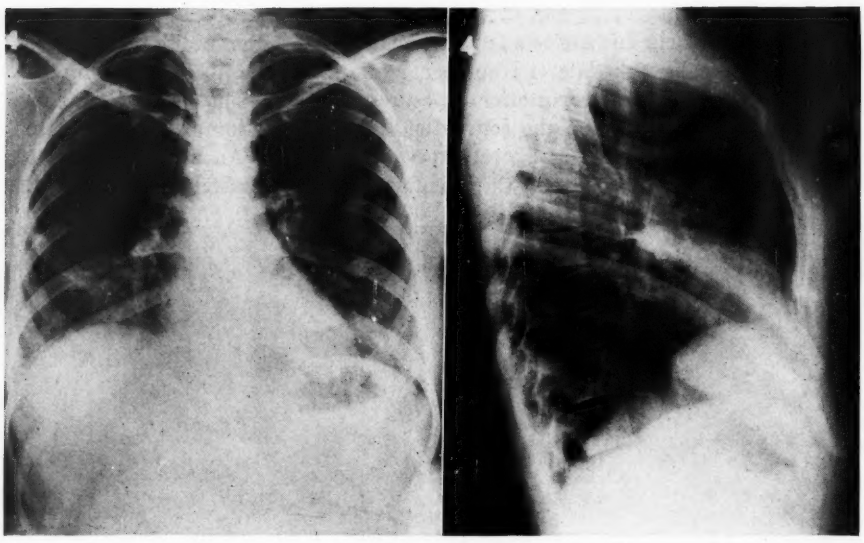
A.

Fig. 2.—Posteroanterior and right lateral roentgenogram of the chest of three different patients illustrating: *A*, the absence of a shadow in the posteroanterior exposure and the evident segmental atelectasis in the lateral exposure; for *B* and *C* see the following page.

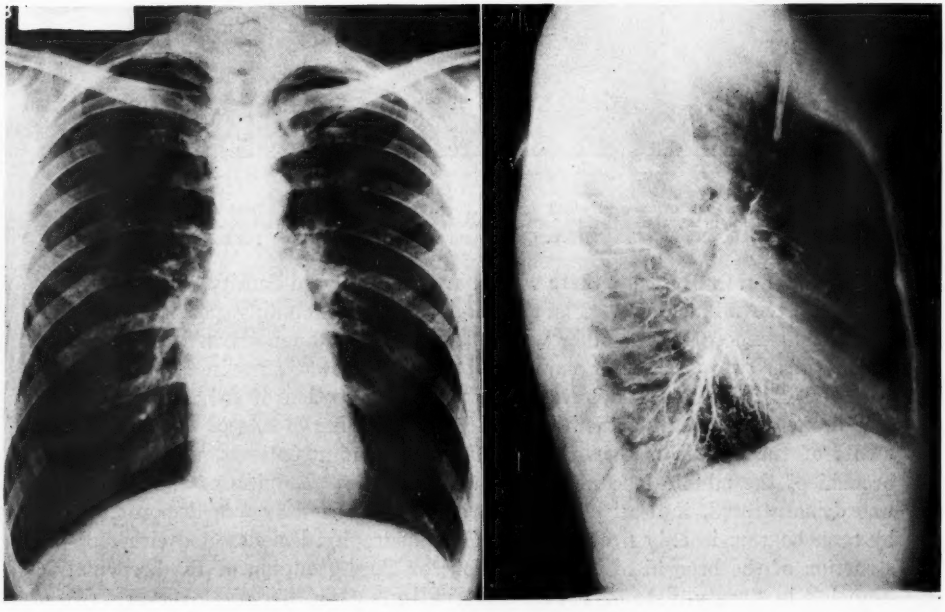
patients and in five patients there was an area of increased density visible in the region of the middle lobe. Plain roentgenograms (including posteroanterior and lateral exposures), in other words, led us to the diagnosis in twenty-four of the thirty-two patients. Complete bronchography was done in twenty-eight patients. The bronchogram revealed complete obstruction of the right middle lobe bronchus in twelve patients and incomplete filling of one of the two segments of the middle lobe in seven patients. In four patients, although the bronchi of the middle lobe were filled with oil, close grouping of the bronchi was demonstrated, indicative of atelectasis. Bronchiectasis was demonstrable by bronchogram in only five patients. Bronchography demonstrated either obstruction of the bronchi of the middle lobe or close grouping of the bronchial branches in twenty-three of twenty-eight patients (Fig. 3).

DIAGNOSIS

The diagnosis of chronic atelectasis and pneumonitis of the middle lobe is based upon a careful history, the bronchoscopic, roentgenographic, and broncho-

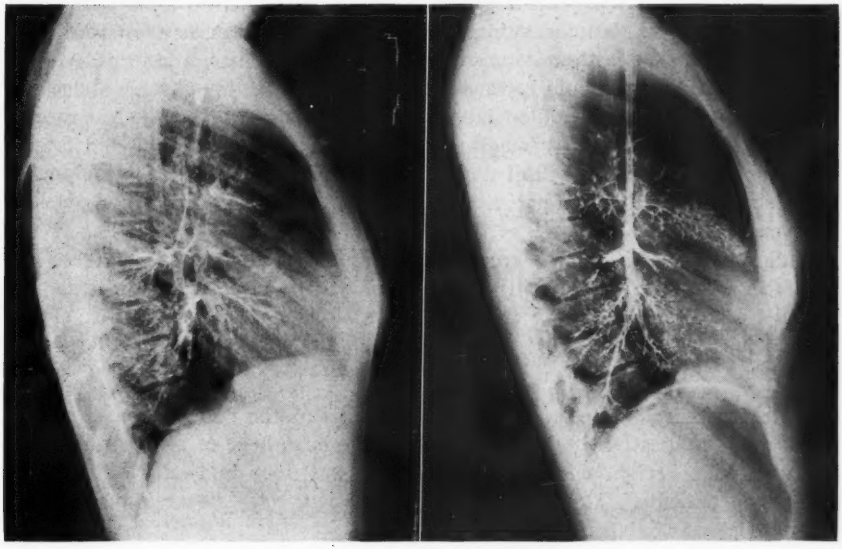


B.

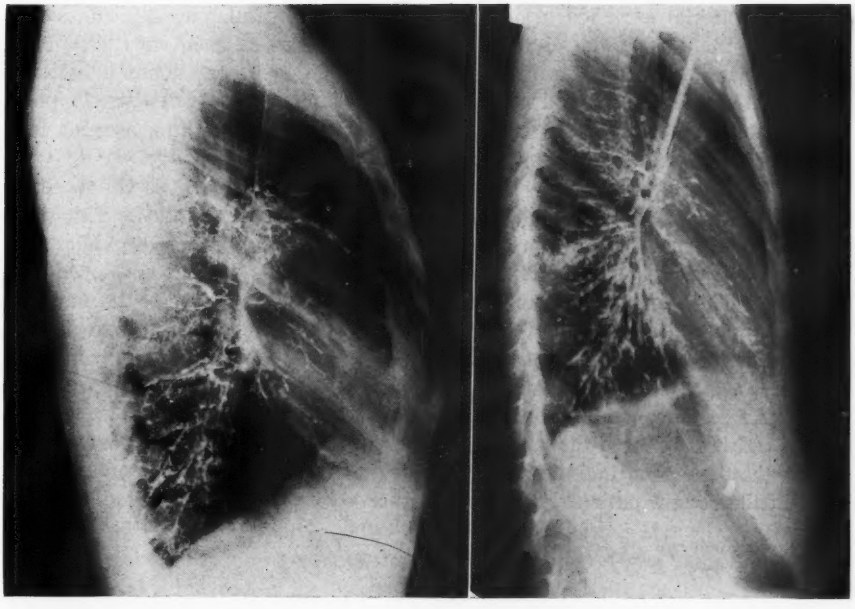


C.

Fig. 2.—For *A* see preceding page; *B*, an area of the middle lobe in the right lateral films, due to calcification within the hilum; *C*, Normal appearance of chest in posteroanterior film but obstruction of bronchus to the middle lobe bronchographically.



A. *B.*



C. *D.*

Fig. 3.—Right lateral bronchograms of four patients illustrating: *A* and *B*, obstruction of middle lobe bronchus; *C*, obstruction of one of the two segments of the middle lobe (the same patient as Fig. 2 *A*); *D*, close grouping of bronchi of the middle lobe.

graphic findings. Any patient giving a history of chronic cough which may be worse in dorsal recumbency accompanied by right anterior chest wall pain and hemoptysis and with a history of recurrent pneumonias, a low-grade fever, and easy fatigability should be considered as possibly having chronic atelectasis and pneumonitis of the middle lobe. Bronchoscopy may suggest the presence of this condition. Plain roentgenograms taken in the posteroanterior and lateral views in many instances will give confirmatory evidence. A complete bronchogram should be made in all patients in order that the remainder of the bronchial tree may be studied as well as the middle lobe. Definite obstruction of the middle lobe bronchi or close grouping of the bronchial branches in those patients in whom the bronchi fill with lipiodol will be visualized in a high percentage of patients suffering from chronic atelectasis and pneumonitis of the middle lobe.

OPERATIVE FINDINGS

Because chronic atelectasis and pneumonitis of the middle lobe is a type of chronic suppurative disease from which there is no hope of recovery, surgery is indicated. A right middle lobe lobectomy was done in twenty-nine of the thirty-two patients. At the time of the operation, complete atelectasis of the middle lobe was found in fourteen patients and partial atelectasis in twelve patients. The lymph nodes surrounding the middle lobe bronchus were unusually large, inflamed, and indurated in fifteen patients. These lymph nodes were calcified in an additional eight patients. In four of these eight patients the calcified material extended into the bronchial lumen. Inflammatory adhesions were present between the visceral and parietal pleura adjacent to the middle lobe in many instances, and in several patients there were dense adhesions between the middle lobe and pericardium and diaphragm. It is entirely possible that the adhesions between the middle lobe and pericardium account for the pain which the patient experiences near the midline anteriorly.

The results of operation have been satisfactory on the whole. Of the twenty-nine patients undergoing a right middle lobectomy, one patient died. This was a 51-year-old man who died on the fourth postoperative day from a pulmonary embolus. There were no postoperative empyemas. One patient developed an organized hemothorax secondary to bleeding. It was necessary to perform a decortication of the remaining portions of the right lung three weeks later. This patient eventually obtained a satisfactory result. Three patients continue to have a slight cough associated with a generalized bronchitis. Two patients have had a recurrence of hemoptysis. One of these patients has a long bronchial stump and it is believed that the bleeding occurs from ulceration of the mucosa within this long bronchial stump. It is believed that the recurrence of hemoptysis in the second patient is due to a rather severe generalized chronic bronchitis.

DISCUSSION

The syndrome of chronic atelectasis and pneumonitis of the middle lobe is presented as a newly recognized clinical entity of pulmonary suppurative disease. Justification for its separation as a clinical entity is occasioned by the

frequency with which this syndrome is encountered together with the characteristic pattern of this type of suppurative disease. It cannot be classified as bronchiectasis. Although bronchiectasis is frequently reported by the pathologists, the principal feature of the disease is obstruction of the middle lobe bronchus with chronic atelectasis and pneumonitis. Although a chronic pneumonitis develops as a result of atelectasis, the condition should not be classified as a lung abscess.

Chronic atelectasis and pneumonitis may occur in any portion of either lung. We believe that various mechanical factors peculiar to the anatomy of the bronchial tree tend to favor the development of bronchial obstruction and atelectasis of the middle lobe more frequently. The right middle lobe bronchus arises from the intermediary bronchus at almost a right angle in the lower portion of the chest. For this reason, drainage is poor. The main bronchus of the middle lobe is also very short and branches after a distance of less than 1 cm. into two main segments. The diameter of these segmental bronchi is normally small. It is entirely possible that as a result of pneumonia there will be inflammation and edema within the wall of these small bronchi. Because of their small diameter, they are easily obstructed. Originating from the remainder of the bronchial tree at almost a right angle, drainage is poor, and atelectasis may readily develop. Two-thirds of the patients in this series gave a history of a pneumonia, almost all of whom had had pneumonia two or more times. It has been our opinion that the first pneumonia perhaps damaged the bronchial wall thus resulting in bronchial obstruction. The lymph nodes of the middle lobe bronchus almost completely surround it. Expansion of an enlarging lymph node within the hilum of the middle lobe is limited medially by the pleural reflection from lung to pericardium in the region of the anterior pulmonary vein and anteriorly by the pulmonary artery as it crosses the bronchus of the middle lobe transversely to its length. As these lymph nodes become inflamed and enlarge secondary to the inflammation within the middle lobe, further pressure is exerted on the bronchus from the outside. If the bronchus is already weakened by inflammation and edema, it is entirely possible that the bronchial wall will be compressed by extrinsic pressure. This then develops into a vicious cycle, the bronchial obstruction inducing more infection within the middle lobe and the infection within the middle lobe inducing further swelling of the enlarged lymph nodes surrounding the middle lobe bronchus. There can be no question of the etiological factor involved in the eight patients in whom enlarged calcified lymph nodes were found surrounding the middle lobe bronchus. In some instances the middle lobe bronchus was surrounded by a ring of calcium. In four patients the calcified nodes had partially eroded through the bronchial wall.

The same set of circumstances may come into play in other portions of the lung, although because of the mechanical factors outlined here, the middle lobe appears to be more commonly involved. Four patients having chronic atelectasis and pneumonitis of the lingular segment of the left upper lobe have been treated

during this same period of time. Three of these patients had an obstruction of the lingular bronchus (bronchographically) associated with enlarged peribronchial lymph nodes, and in one patient the obstruction was due to a calcified lymph node. Chronic atelectasis and pneumonitis of the right upper lobe due to enlarged calcified lymph nodes about the right upper lobe bronchus were encountered in one patient.

During the last three years we have done 168 pulmonary resections (exclusive of total pneumonectomies) for various types of pulmonary inflammatory disease. Twenty-nine of these were done for chronic atelectasis and pneumonitis of the middle lobe. Exclusive of lung abscess, bronchiectasis, and specific types of pneumonitis, chronic atelectasis and pneumonitis of other portions of lung constituted the indication for resection in twelve patients.

Graham, Burford and Mayer,* presented a paper entitled "The Middle Lobe Syndrome" before the Interstate Postgraduate Assembly in October, 1947. At that time they presented twelve cases and also were of the opinion that the middle lobe syndrome justified separation as a clinical entity.

The diagnosis of chronic atelectasis and pneumonitis of the middle lobe is not a difficult diagnosis to make if one is aware of the possibility. The most important condition in its differential diagnosis is bronchiogenic carcinoma. In all instances in which a preoperative diagnosis of chronic atelectasis and pneumonitis in the middle lobe is made, the possibilitiy of a bronchiogenic carcinoma must be kept in mind, particularly in patients in the older age group.

It is believed that many patients complaining of chronic cough, chest pain, and hemoptysis have had only plain posteroanterior roentgenograms made and given a diagnosis of bronchitis. Such a patient should have the benefit of a lateral roentgenogram and a complete bronchogram. A careful history and complete bronchography will reveal signs of middle lobe obstruction in some of these patients. Even though a complete bronchogram be done, lack of filling of the middle lobe bronchus is too often easily explained on the basis of technical failure. If there is any question concerning technique, the bronchogram should be repeated. If as a result of a second bronchogram, oil does not enter the bronchus of the middle lobe or one of its segments, a positive diagnosis of bronchial obstruction should be made. The procedure, of course, necessitates doing complete bronchography with careful attention to all bronchial segments

The treatment of chronic atelectasis and pneumonitis of the middle lobe is surgical. The mortality and morbidity in this series were low. Because of the possibility of a bronchiogenic carcinoma, surgery is imperative in the patients in the older age group. Pulmonary emphysema and an obstructive bronchitis will probably develop if the localized area of atelectasis and pneumonitis is allowed to persist over many years. It is our opinion that as soon as it becomes obvious that the atelectasis and pneumonitis are chronic a pulmonary resection should be done.

*Graham, Evarts A., Burford, Thomas H., and Mayer, John H.: The Middle Lobe Syndrome, Postgrad. Med. 4: 29-34, 1948.

SUMMARY

A review of thirty-two patients having chronic atelectasis and pneumonitis of the middle lobe is presented. The cardinal symptoms of this condition are cough, anterior chest wall pain, hemoptysis, dyspnea, and wheeze. A history of recurrent pneumonias, low-grade fever, easy fatigability, frequent colds, and loss of weight is obtained in a high percentage of patients. Pathologically the condition is characterized by chronic atelectasis and pneumonitis of the middle lobe secondary to bronchial obstruction. The bronchial obstruction is the principal etiological factor in the development of the chronic atelectasis and pneumonitis. The frequency and the characteristic diagnostic pattern of this type of pulmonary suppurative disease justifies its separation as a clinical entity.

DISCUSSION

DR. JOHN H. MAYER, Kansas City, Mo.—We too have been impressed by the increasing number of patients with chronic nontuberculous atelectasis of the right middle lobe. Drs. Graham, Burford, and I have had the opportunity recently to report our experience in thirteen such cases. The symptomatology, x-ray, and bronchoscopic findings have been essentially the same as in the cases reported here today. Hemoptysis has been the presenting complaint in the majority of our patients. We are in complete agreement with Dr. Paulson as to the importance of the lymph nodes about the middle lobe bronchus in effecting constriction of that bronchus. The strategic location of these nodes and their importance in the etiology of nontuberculous bronchiectasis have been emphasized by Brock in his recently published book on the anatomy of the tracheobronchial tree. (Slide.) This illustration was taken from his book and demonstrates, as Dr. Paulson has told you, the location of the lymph nodes about the origin of the right middle lobe bronchus.

Because these cases began to make their appearance at a time when penicillin was coming into widespread use it occurred to us that these patients might represent the residua of extremely severe acute suppurative infection from which the patients would not have recovered except with the help of penicillin. However, in reviewing our cases we found that many of them had never received penicillin and most of them had not had a severe acute onset. Our concept is that no one specific etiological agent can be indicted, but that any disease producing pulmonary lymphadenopathy may be at fault.

I think it may be worth while to point out that these lymph nodes represent to a large extent lymph drainage from the lower lobe on the right side and that one must be careful that the right lower lobe is free of disease prior to attacking the middle lobe surgically. In one of our patients we subsequently had to remove chronic suppurative disease of the right lower lobe, the right middle lobe having been successfully removed six or eight months previously.

DR. RALPH A. DORNER, Des Moines.—I have seen a number of patients who fit into this syndrome. I would like to mention one patient who presented a very typical clinical and x-ray picture. While operating on her after freeing the hilus of the middle lobe, I noticed that the bronchus to the middle lobe was very thick. Previously, the bronchoscopy had shown a comma-shaped narrowing of the bronchus to the middle lobe. I was afraid, when we found this thickening of the bronchus, that perhaps something had been missed on the bronchoscopy. I, therefore, opened the bronchus and, to my amazement, a peanut fell from the bronchus. There was a nest of granulation tissue around the nut accounting for some of the thickening of the bronchus and the narrowing of its orifice. Thus the point was brought to mind that even though the picture may fit the syndrome which Drs. Graham, Burford, and Mayer described earlier, we must consider that patients may have foreign bodies within the bronchus producing a clinical picture of the "middle lobe syndrome."

DR. ARTHUR S. W. TOUROFF, New York.—I have observed, and operated upon, a number of cases similar to those reported by Dr. Paulson and Dr. Shaw, and I am in agreement with practically everything that they have said. I have been impressed with the difficulties that may arise in connection with making a diagnosis in older patients, and especially with ruling out the diagnosis of malignancy. Carcinoma of the middle lobe is not particularly common; and yet if the patient is in the so-called "cancer age" and has hemoptysis and clinical symptoms of pulmonary infection, as well as roentgenographic evidence of atelectasis, one cannot dismiss lightly the diagnosis of carcinoma. Nevertheless, the presence of calcific glands should always make one suspicious of the presence of a nonmalignant lesion. It is interesting to note that in a number of our cases, bronchoscopy has been entirely negative and that, under such circumstances, bronchography has been of great value in clarifying the diagnosis. In such cases, the stenotic area is situated too far distally in the middle lobe bronchus to be seen by the bronchoscopist. If lipiodol is instilled into the bronchus, it is not uncommon to note, in the bronchogram, an area of bronchial narrowing which may be one-half inch or more in length. This is in distinct contrast to the complete occlusion of the bronchus or the short area of bronchial narrowing that usually is seen in cases of malignancy. In certain cases, it may be quite impossible to rule out the diagnosis of malignancy prior to operation, and under such circumstances, surgical exploration rather than "watchful waiting" is indicated. Furthermore, in certain cases even after the thorax has been opened, it may be impossible to differentiate between a chronic inflammatory lesion of the middle lobe and a malignancy. In a number of cases of this type, I have been forced to rely upon the result of frozen-section examination; for that reason, it is well to have the pathologist present in the operating room whenever the diagnosis has not been made with certainty prior to operation. This simple expedient has, on several occasions, permitted us to rule out the diagnosis of malignancy and proceed with simple removal of the middle lobe rather than pneumonectomy.

DR. CLARENCE CRAFOORD, Stockholm.—It might be of interest to you to hear that it is not only in Anglo-American countries that cases of this type occur. We have had a number of them also in Sweden. It is to one of the symptoms described here that I want to call attention, that is, hemoptyses. We had one case with very large hemoptyses, and we did not believe that the small middle-lobe lesion that we found in the roentgenogram could be the cause of the abundant bleeding that was present. The patient had had for about 15 or 20 years smaller hemoptysis at intervals, sometimes at intervals of years, but in the last few years he had had a number of hemoptyses so great that he went into unconsciousness at the time of bleeding; the blood loss each time was estimated to be more than 1 liter. At these occasions he was taken to a provincial hospital and when the bleeding had stopped the secondary anemia was treated, after which he felt fairly well and went back to his work. Finally he was sent to us and bronchoscopy was done; when the right main bronchus was entered enormous bleeding began and the patient nearly died. The bleeding, however, could be controlled and localized to come from the middle lobe bronchus on the right side. At operation, the chronic inflammatory reaction in the area of the hilum was such that I encountered technical complications so I had to perform a total pneumonectomy. The specimen afterward showed a very peculiar thing; there was a fistula as big as a slate pencil between the pulmonary artery branch of the middle lobe and the bronchus, and in that hole was a thrombus. We believe that the origin of this fistula must have been a purulent lymph node inflammation with an abscess breaking into both the bronchus and the pulmonary artery.

DR. JEROME HEAD, Chicago.—I believe that we are making a mistake in limiting our discussion to involvement of the middle lobe. The same process, of course, may affect any lobe. As you know, Parrott is remembered because he said that in tuberculosis the hilus lymph glands reflected the lung parenchyma. When I started tuberculosis work, it was common for the roentgenologist to put his hand over the hilus region and say, "Forget about that area; if you are looking for tuberculosis, look in the parenchyma." Increasingly

I have been impressed with the importance of looking to the hilus. Very often parenchymal disease is a reflection of trouble in the root glands. This is true both in tuberculous and in nontuberculous disease. We are seeing today more primary progressive tuberculosis. I am certain that in many of these cases severe bronchial tuberculosis develops by extension from involved bronchial lymph glands, and also that extension occurs from these glands into the parenchyma.

Our attention has recently been called to this whole subject of the glands in the mediastinum and the root of the lung by an increasing series of cases in which patients have expectorated calcium. At the meeting in New York in two weeks, Dr. Chester Moen and myself are giving a paper on nontuberculous pulmonary disease secondary to nontuberculous reinfection of calcified glands at the hilus. In our series, this has affected all lobes. Dr. Moen has made 100 dissections in the anatomic laboratory and has found, as other people have found, that the location of these glands is very constant. I shall not go into the details of the anatomy, but would like to mention that Mr. Nelson, whom many of us remember, was one of the first to work out the detailed anatomy of the lymph glands of the hilus of the lung. He was mentioned yesterday in connection with original anatomic work on the segments of the lung lobes.

DR. ALFRED GOLDMAN, Los Angeles.—Many points have been covered regarding this disease. I became interested in it about two years ago and I find that once you begin to look for the effects of calcification in x-ray views of the chest you begin to explain almost all the previously unexplainable symptomatology on the basis of this calcification. As a result, sometimes I think we overdo it, because it is sometimes hard to tell which came first, the lymph nodes or the disease in the lung, and I still am confused myself to know in some cases whether there was bronchiectasis and then the lymph nodes, or whether the lymph nodes came first and bronchiectasis followed. I think that I agree with Dr. Graham and others who feel that the lymph nodes were the primary process in many instances.

(Slides.) Also I agree that this is a disease that requires special attention particularly in the older age group to differentiate it from carcinoma.

This is one of six cases; the base of the right upper lobe was involved. The patient was about 50 years of age, had a small hemoptysis, and it was thought she had carcinoma. Bronchoscopy showed narrowing and bulging and a diagnosis of suspicion of carcinoma was made. I performed exploratory thoracotomy with a diagnosis of carcinoma undetermined. A right upper lobectomy was done.

I urge that when exploration is carried out in such patients not only the pulmonary calcification be taken out but others searched for which may cause further trouble; the chances are that the calcifications produced the hemoptysis in this case. There was also an empyema which produced a large amount of pus in the interlobar space.

It will be noted that the large paratracheal calcification was removed as well as that one penetrating toward the lumen of the right upper lobar bronchus.

This patient made an uneventful recovery and has remained well for the past 1½ years.

DR. DONALD L. PAULSON, Dallas.—We are naturally pleased that others are seeing this condition as well as ourselves. In talking to several men in the last few days they have told us that they have encountered similar cases. As pointed out to us on Monday the term "obstructive pneumonitis" is preferable to the term "chronic atelectasis and pneumonitis." I believe this is particularly true in this group of cases under discussion this morning.

In regard to foreign bodies within the bronchus, at least two patients in this series had a foreign body reaction in the bronchial wall, both being due to strands of cotton, one in a 53-year-old woman with symptoms of twenty years' duration, and the other in a 7-year-old boy.

We agree that carcinoma is the most important condition to be considered in the differential diagnosis particularly in patients in the older age group. Surgery, of course, is imperative in that group for this reason. In our experience in only one instance have we made a diagnosis of chronic atelectasis and pneumonitis of the middle lobe and found a carcinoma at the time of the operation.

We certainly do see this condition in any portion of either lung. There is nothing specific about the pathology except that it does represent an obstructive pneumonitis. In our experience it does occur much more frequently in the middle lobe and perhaps this is due to the mechanical factors outlined in our discussion here this morning. Because of the frequency with which an obstructive pneumonitis occurs in the middle lobe and because of the characteristic clinical and diagnostic pattern, we believe that this syndrome is worthy of separation as a definite clinical entity.

DR. PAUL C. SAMSON, Oakland, Calif.—Dr. Dugan and I wish to record our experiences with the "middle lobe syndrome." Our interest in these lesions was heightened by discussions with Dr. Thomas Burford at the St. Louis meeting of this Association and by perusal of Brock's excellent monograph. In the past eighteen months, we have cared for seventeen patients with localized bronchiectasis and/or pneumonitis of the right middle lobe. Seven additional patients with bilateral bronchiectasis (in all of whom left-sided resections have been performed) showed, on the right, bronchiectasis only of the middle lobe. During the same period of time we have seen but three patients whose bronchiectasis was limited to the lingula, and three whose bronchiectasis was limited to the anterior segment of the right upper lobe. Of the seventeen patients noted here, fifteen were women and two were men. The majority of our patients with bronchiectasis have been women in the past two years but apparently there is an increased and as yet unexplained sex incidence in the middle lobe group. With the exception of one girl of 19 years whose symptoms dated from babyhood, the remainder first had difficulties in adult life and the average age of the sixteen patients was 51 years (30 to 69 years). Hemoptysis was the presenting symptom in ten; cough, sputum, and febrile episodes in seven.

Ten patients showed bronchiectasis primarily (six with atelectasis) on the basis of bronchograms and surgery; seven patients (four with atelectasis) showed no bronchiectasis following lipiodol instillation. The right middle lobe was resected in five patients; surgery was recommended to another five patients and presently refused (probably until bleeding begins again); in the remaining seven excision was not advised because of age, and improvement following bronchoscopic aspiration and antibiotic therapy.

Of the five patients on whom resection was performed, three showed definite evidence of partial compression of the middle lobe bronchus because of enlarged lymph nodes and in two there was no evidence of bronchial compression.

INTRACAVITARY SUCTION (MONALDI) IN THE TREATMENT OF EMPHYSEMATOUS BULLAE AND BLEBS

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BULLOUS emphysema is a relatively common and often a seriously crippling condition. The etiology and diagnosis of emphysematous changes in the lungs have received considerable attention in the literature, but the treatment, until very recently, has been seldom mentioned and uniformly considered hopeless.

In 1687 Bartholinus[1] first described a large pulmonary cyst in a 4-year-old boy who died of "marasmus." Sir John Floyer,[2] in 1698, described the cystic alterations in the lungs of broken-winded horses, and his observation would be equally applicable to human beings. Laennec,[3] however, was the first to put pulmonary emphysema upon the list of recognizable diseases and catalogue its symptoms and pathologic changes. Laennec stated that Ruysch and Valsalva had observed the dilatation of pulmonary cells and that Morgagni clearly described this dilatation.

From a review of the literature, there appears to be no uniformity of nomenclature or opinion on the differential diagnosis of cystic lesions of the lung. The terminology apparently depends upon which portion of the clinical or pathologic picture the author is describing.

Congenital pulmonary cysts, congenital and acquired bronchiectasis, epithelial lined cavities following pulmonary suppuration, lung abscess, tuberculous cavities, cavities due to fungus or parasitic infections, spontaneous pneumothorax, and the various types of emphysematous changes of the lungs must all be considered in the differential diagnosis of so-called pulmonary cysts.

Cases of emphysema with emphysematous bullae and blebs have come to our attention with increasing frequency. It is the purpose of this paper to present the results of surgical treatment by intracavitary suction in nine cases showing these emphysematous changes in the lungs.

PATHOGENESIS

The etiology of emphysema is uncertain and its pathogenesis remains subject to extensive investigation. For the purpose of definition, Christie[4] classified emphysema under five types: hypertrophic, or chronic obstructive emphysema; atrophic, or senile emphysema; acute vesicular emphysema associated with acute infections of the respiratory tract; localized, or compensatory emphysema due to atelectasis or fibrosis; and surgical, or acute interstitial emphysema associated with chest injuries or artificial pneumothorax. Of these types, chronic obstruc-

From the surgical services of Hines Veterans Administration Hospital and Northwestern University Medical School.

Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

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tive emphysema is probably the most important in the formation of bullous emphysema, although any type could contribute to the check valve mechanism which produces bullae or blebs.

The terms "bullae" and "bleb" are used in this paper as described by Miller.[5] Blebs as well as bullae represent localized air pockets within the lungs. Blebs are located immediately beneath the pleura, while bullae are within the lung tissue itself. In the case of the bleb, air escaping from a ruptured alveolus leaks into the subserous connective tissue layer of the pleura which is composed of collagenous and elastic fibers. The escaping air separates the pleura from the underlying alveoli. He noted advanced emphysema in all cases in which blebs occurred. Bullae are also caused by the rupture of alveoli, but the atrophic and dilated alveoli rupture into each other forming air pockets within the parenchyma of the lung with the pleura retaining its connection with the lung.

Bullae usually occur in patients with chronic bronchitis or asthma, but they are occasionally seen in individuals with no demonstrable respiratory disease. One can speculate that in chronic bronchitis, the infection causes swelling of the walls of the bronchi, and this together with the thick tenacious secretions causes obstruction to free gaseous interchange. This valvular mechanism was found by Hayashi[6] on microscopic examination of bronchi communicating with emphysematous cavities. Spasm of the bronchial muscles in asthma has long been recognized as an important factor in the bronchial obstruction of this disease. Vesicular emphysema with subsequent bulla formation is often seen following a severe pneumonia[7] or lung abscess,[8] where there is necrosis of the alveolar walls. McNeil[9] in studies of pneumonia in children found that 20 per cent of the cases autopsied showed necrosis of the bronchial and bronchiolar walls. This necrosis might lead to rupture and interstitial emphysema, which could dissect into the subpleural layer to form blebs. With these similar pathologic processes occurring in the same area of the lung, rupture and fusion of the blebs and bullae may take place to form huge so-called pneumatoceles.

Compensatory emphysema is seen in atelectasis, and following lobectomy or pneumonectomy.[10] It is also a contributing factor in so-called "senile emphysema" where the chest becomes barrel-shaped with increased thoracic volume.[11, 12]

Normally, the active force of inspiration is greater than the passive action of expiration, but the elastic recoil of the lung and the changes in dynamics of the thorax deflate the unobstructed portions of the lungs. In partial obstruction of the bronchi, a volume of air is drawn into the lung but only a portion of air escapes, thereby creating increased intra-alveolar tension with subsequent stretching, atrophy, and rupture of the walls.[13]

In our studies of bullae through the thoracoscope, we have learned that once tension cavities have formed, the dynamics of their progression are reversed so that the destructive force is now that of expiration instead of inspiration. These large emphysematous cavities are at least partially surrounded by elastic and compressible lung parenchyma, and they are supplied air by tortuous, distorted bronchi. A typical bullous cavity appears as a large air-filled sphere, the base of which is made up of long slender, interlacing strands of black pulmonary tissue. The dome of the cavity is covered with a thin, white membrane of pleura,

crisscrossed with a fine capillary network. Through this translucent membrane the rib cage can be seen sliding back and forth with respiration, apparently because of the absence of pleural adhesions. Long filaments of tightly stretched lung tissue may be tented from the walls to the floor of the cavity. During expiration, as the intrathoracic pressure increases, the pressure within the lung becomes greater than within the bulla. The parenchyma can be seen to bulge into the cavity toward the scope where many minute bronchial openings can be seen to appear among the strands of tissue. At the height of expiration, with the tip of the scope near the openings, the lens becomes fogged by the incoming moist air.

During inspiration, as the intrathoracic pressure decreases, the walls of the cavity expand because the pressure within them is greater than in the remainder of the lung. The parenchyma can be seen to recede from the scope, and the pores disappear as the web of lung fibers become compressed on one another. The tortuous bronchial openings to the cavity become more obstructed as the surrounding parenchyma is compressed by the expanding cavity, and air is thus trapped within the bulla. Coughing or straining against the closed glottis exaggerates this check valve mechanism so that pressures within the cavity have been measured as high as plus 50 mm. of mercury. Blebs present essentially the same picture except that the major portion of the cavity wall is made up of thin pleura. There is a smaller area of denuded lung, and there are fewer bronchial fistulas than are usually seen in bullae.

It is apparent that emphysematous changes in the lungs are due to complex processes. Any one or all of the afore-mentioned factors, as well as many unknown mechanisms, may contribute to the formation of bullae and blebs.

Emphysema is found in 2 to 5 per cent of all autopsies.[4] It seems probable that bullous emphysema may become a more pressing medical problem in the future, as the antibiotics permit more persons to survive severe respiratory infections, and as more people live into the later decades of life.

CLINICAL PICTURE

The symptomatology depends upon the underlying pathologic processes and the resultant physiologic changes. Because of the nature of emphysema, bullae and blebs usually are multiple, often are bilateral, and symptoms tend to progress. Although some cases are entirely without symptoms and are discovered on routine chest x-ray examinations, the majority of patients' complaints are referable to chronic bronchitis or asthma, which are known to be associated with emphysematous changes. The presence of a large air space, essentially nonfunctional, can cause dyspnea and cyanosis by replacing space normally occupied by functioning lung. The total capacity, as well as the vital capacity, is reduced.[14] The thorax may increase in size on the side of the lesion and the diaphragm become depressed to cause further mechanical impairment of the respiratory bellows. The intercostal spaces are widened, and the affected side lags on inspiration. The belbs or bullae may reach sufficient size to cause mediastinal shift and circulatory embarrassment. It is not uncommon for these patients to die of anoxia.

Chest pain is not infrequent and may be due to tension or to adhesions caused by readjustments of the lung due to pressure from bullae.

Infection in bullae and blebs is extremely rare, in contrast to the frequency with which this complication is seen in other types of pulmonary cysts. We have seen only two cases where this developed. These were apparently due to active inflammatory processes in the lung immediately adjacent to the bullae.

Emphysema and decreased respiratory reserve lead to obstructed pulmonary circulation,[15] high pulmonary arterial pressure, and hypertrophy of the right side of the heart.[16] With cor pulmonale the peripheral venous pressure increases,[17] and the patient with far-advanced bullous emphysema may present himself with the symptoms of right heart failure.

Spontaneous pneumothorax is by far the most frequent and dangerous of the sequelae. It is often recurrent and is the most common cause of death in the cases of bullous emphysema reported in the literature.[18-26] Leach[27] has recently demonstrated that the etiology of spontaneous pneumothorax is due almost entirely to the rupture of emphysematous blebs and bullae, and only rarely due to tuberculosis, which was once thought to be the major cause of this complication.

X-RAY FINDINGS

Blebs and bullae appear on the x-ray films as areas of decreased density or increased brilliancy. They cause annular, ring-shaped shadows with fine, hair-line borders which are in contrast to the heavy borders seen in inflammatory excavations.[28-32] A slight haziness is often noted around a smaller bulla, and above or below a larger bulla, due to compression atelectasis of the adjacent parenchyma. The bullous spaces may be crisscrossed with lacy filaments of lung tissue. In advanced bullous emphysema the tension cavities which produce the so-called "vanishing lung"[33-34] may be difficult to differentiate from complete pneumothorax. The heart and mediastinum may be displaced toward the normal side, and the diaphragm on the affected side depressed and flattened. In pneumothorax the elasticity of the lung causes it to contract around the hilus, forming a rounded stump. However, with large bullae the hilar shadow appears narrow and elongated by compression, or may not be seen at all, and the lung can be pushed peripherally where its shadows may be seen in the apex or costophrenic angle. Furthermore, in spontaneous pneumothorax there is likely to be fluid in the pleural cavity. Another differential clue was pointed out by Korol,[35] who noted that there was a narrowing of the intercostal spaces due to the decreased intrapleural pressure of chronic pneumothorax, whereas there was a widening of the rib cage in the expansile cavities of emphysema.

It is important to remember that the routine chest film often fails to reveal emphysematous cavities. This is particularly true where the cavities are in the base of the lung. In this location their contours may be covered by bronchial markings of lung which may lie in front or in back of the cavity. Where bullae or blebs are suggested, it has been our practice to take several oblique and lateral views. The films are exposed a few kilovolts lighter than normally calculated because of the decreased density of these lungs.

RATIONALE OF TREATMENT

Emphysematous bullae and blebs require treatment because they are causing recurrent or chronic pneumothorax or because they are compressing the lung and causing incapacitating dyspnea.

The surgical attack may be directed toward release of pressure and the reversal of the check valve mechanism by drainage, the closure of the bronchial communications, or the excision of the diseased portions of the lung.

Early attempts at treatment of tension cavities by thoracentesis gave only temporary relief of symptoms[36-39] and was often followed by shock, tension pneumothorax, and death.[40, 41]

Lilienthal[42] in 1929 reported the alleviation of symptoms of a tension cyst by temporary tube drainage with the use of a one-way valve. Croswell[43] reported another case of giant cyst treated in a similar manner. The cyst collapsed to about one-fourth its original size, and the patient's clinical symptoms disappeared. Brown and Brock[44] treated a large "balloon" cyst by continuous suction drainage with gradual collapse of the cavity. Drainage was continued by making an Eloesser endocutaneous flap[45] until the lung was completely re-expanded.

Thoracoplasty was suggested[46] and used in a few cases without beneficial results. Chondrectomy was proposed by Freund[47] to relieve the intrathoracic pressure seen in emphysematous changes of the lung. This procedure failed as it obviously ignored the basic pathology and allowed additional expansion of the lung.

Lipiodol injected into the cavities was sometimes followed by relief of symptoms. This may have been due to blocking of the bronchial fistulas with oil or by inflammatory changes.[43, 48, 49] Sclerosing solutions have also been suggested.

Transthoracic closure of bronchial fistulas in tension cysts was described by Wood in 1934.[49] Recently Naclerio and Langer[50] reported an additional four cases treated in a similar manner where they used the walls of the bulla or bleb to cover the defect in the lung. We have treated one case by excising the roof of the bleb and re-expanding the lung with suction drainage.

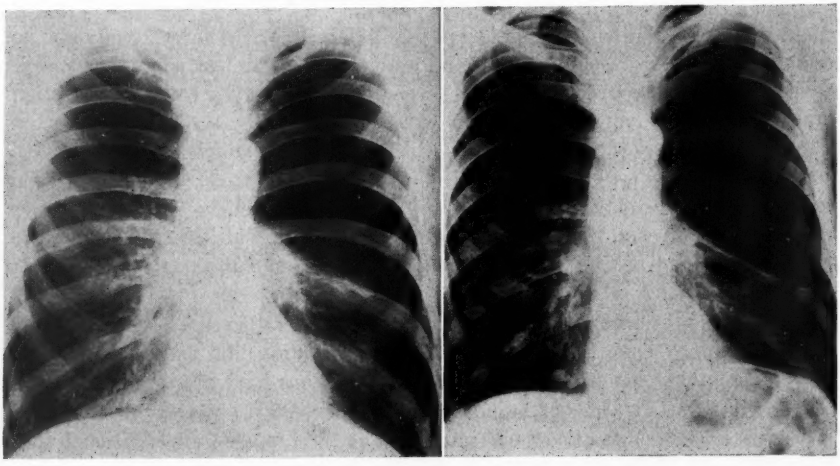
Excision of bullae is not surgically possible because there is no epithelial lining or cleavage plane. However, segmental resection, lobectomy, or pneumonectomy may be performed if the disease is not too extensive or bilateral. Recurrent pneumothorax and chronic tension pneumothorax, due to rupture of blebs or bullae, often force the hand of the surgeon to exploratory thoracotomy. The method of treatment then depends upon the findings, which often cannot be accurately predetermined until the chest is opened. Tyson,[51] Meade,[52] and Clagett[53] have used this approach successfully.

Transthoracic exploration should be avoided, if possible, as thoracotomy alone may produce inflammatory changes of the lung and pleura that may seriously impair vital capacity which may already have reached minimum functional tolerance.[54]

Because some of our patients with large bullae were in such critical condition, major surgery could not be undertaken, and it was felt that intracavitary drainage was the only procedure that they would tolerate. Our initial efforts

met with such success that we have now come to regard it as the procedure of choice in most cases.

The point of optimum drainage is chosen by studying the various x-ray views and selecting a site where the bleb is adjacent to the chest wall. Under local anesthesia, an extrapleural exploration is done to determine whether or not there is pleural symphysis. In every case to date a free pleural space has been found, and it has been necessary to produce adhesions with gauze packing before the catheter could be inserted. The constancy of this finding suggests that the absence of pleural adhesions is necessary for the formation of the blebs. It seems probable that the inflammation which produces adhesions fixes the visceral pleura so that it cannot be separated.



A. *B.*

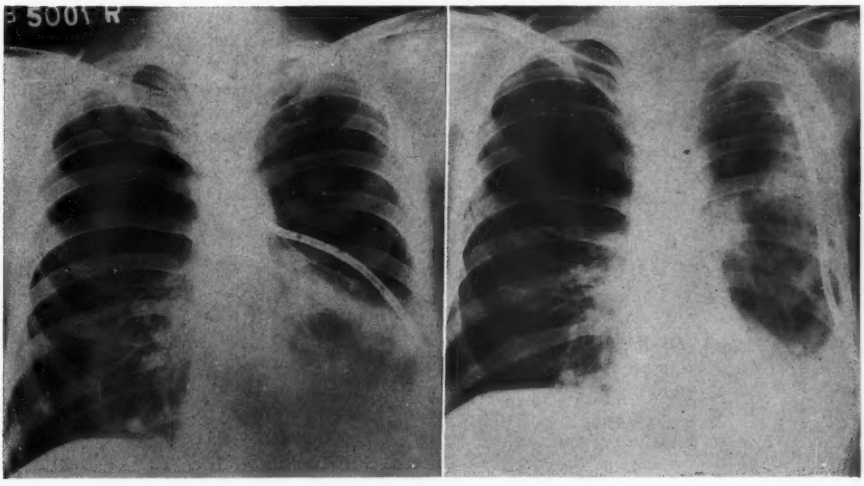
Fig. 1, *A* and *B* (Case 1).—*A*, Chest x-ray film taken July, 1945, showing absent lung markings in upper two-thirds of left side of chest and beginning changes in right apex. *B*, Film of December, 1946, showing progression of disease.

The gauze pack is removed as a second stage procedure, and the wound is allowed to heal before proceeding with the catheter insertion. Pressure readings are then recorded, a thoracoscopic examination is done, and biopsies are taken, if feasible. A multiple fenestrated catheter is then placed well into the cavity, sealed at the skin edges with an airtight dressing, and connected to a water-seal bottle. If air continues to escape through the bottle and the lung has shown no evidence of re-expansion after a few days, a Stedman suction pump is added and adjusted to maintain a negative pressure of minus 10 to 20 cm. of water. Re-expansion has usually occurred in one to three weeks. The catheter is allowed to remain in place an additional few days to cause inflammatory obliteration of the space. The mechanism of re-expansion is believed to be twofold; the vicious cycle of events causing a check valve tension cavity is broken by substituting a gentle, physiologic, negative pressure which allows the re-expansion of surrounding, compressed, atelectatic lung; the catheter acts as a foreign body causing

effusion and inflammatory closure of the bronchial fistulas. Gross infection, which was a stumbling block to this type of treatment in the past, can now be controlled with antibiotics.

It should be pointed out that this type of treatment is difficult because it requires considerable time, meticulous attention to details, and persistence in drainage to obtain re-expansion of the lung. The results, to date, have been gratifying, but it will be necessary to carry out a long follow-up study on these patients to determine the outcome of this type of procedure.

Intracavitary suction drainage does not give satisfactory results in epithelial lined cysts which are better treated by excision. We have inadvertently drained this type of cyst in the past. In future cases, where the differential diagnosis is not possible preoperatively, we feel that careful thoracoscopic examinations should be performed, and biopsies taken if necessary, before the ultimate type of th rapy is decided.

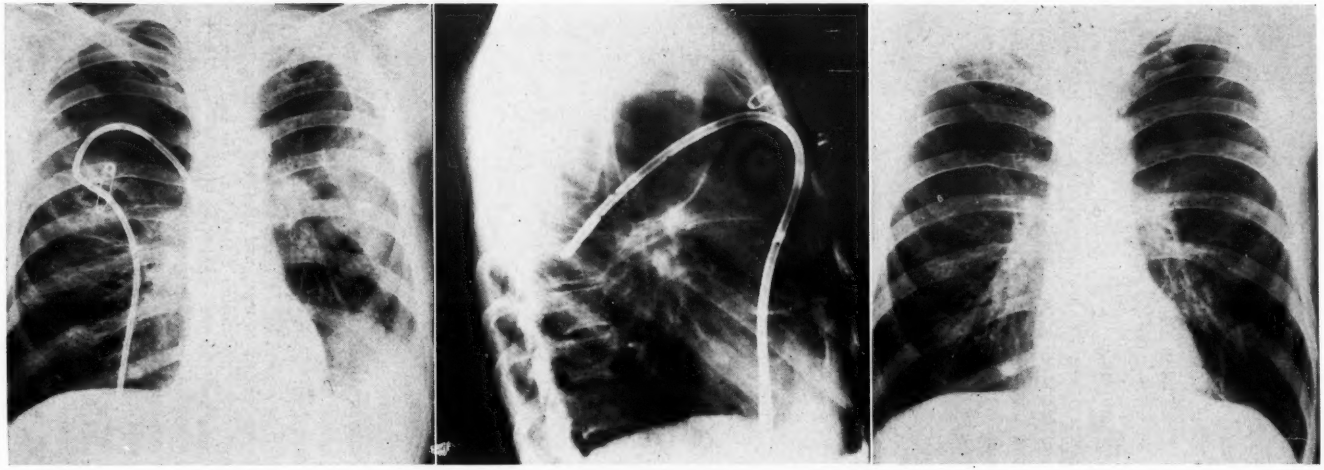


C. *D.*

Fig. 1, *C* and *D* (Case 1).—*C*, Catheter in left bullous cavity. *D*, Bullae decompressed; left lung expanded.

CASE REPORTS

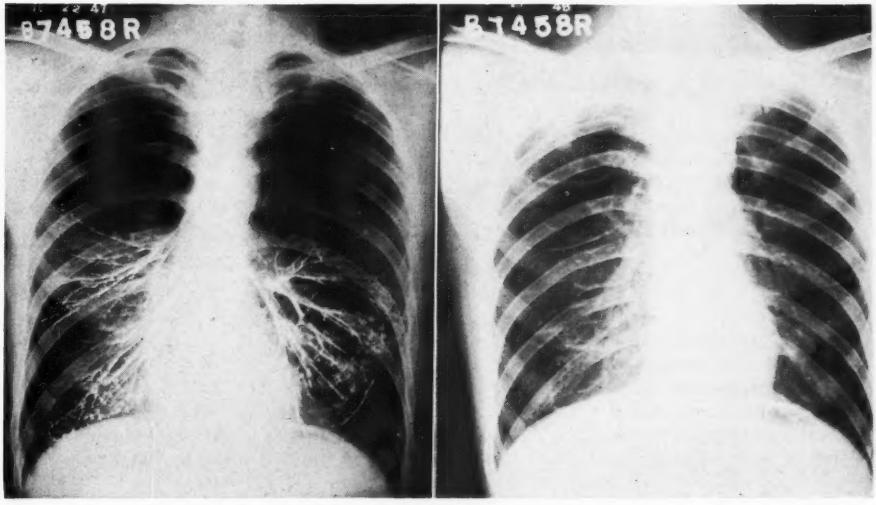
CASE 1.—R. G., a 50-year-old white man, was admitted January, 1947, with a history of chronic severe dyspnea and acute abdominal distention. He had been previously hospitalized for treatment of asthma and chronic appendical abscess. The patient stated that the asthma had become progressively worse during the past five years and that he had a constant cough productive of small amounts of thick, sticky sputum. He was bedfast because of dyspnea and said that he had been taking 10 to 20 c.c. of adrenalin daily by nebulizer to help him "reach for his wind." Hospital work-up revealed a partial intestinal obstruction and far-advanced bullous emphysema. In a review of x-ray films taken July, 1945, there was noted absence of lung markings in the upper half of the left side of the chest and a slight decrease in markings in right upper lung field (Fig. 1 *A*). The film of



E. *F.* *G.*

Fig. 1, *E*, *F*, and *G* (Case 1).—*E*, Catheter in right bullous cavity. *F*, Lateral view showing catheter in bulla. *G*, Postoperative film with both lungs re-expanded.

the present admission showed progression of the disease so that lung markings were now absent in the upper three-fourths of the left side of the chest and the upper half of the right side of the chest (Fig. 1 *B*). At exploratory laparotomy adhesions were cut to relieve the small bowel obstruction. After a stormy postoperative course, the patient was transferred to the Thoracic Surgery Service where it was decided that intracavitary suction was the only possible form of therapy that the patient might tolerate. The vital capacity was 950 c.c. The usual three-stage Monaldi procedure was carried out on the left side, as it was necessary to produce pleural symphysis before insertion of the catheter. Intracavitary pressure readings were recorded at plus 10 minus 8 mm. Hg on quiet expiration and inspiration. With coughing the needle swung to plus 50 (as high as the machine registered) and back to minus 20 on deep inspiration. Thoracoscopy revealed a huge bullous cavity similar to a picture of these cavities described previously. Biopsy of the wall of the cavity revealed inflammatory tissue, pleura, and a few distorted alveoli. The diagnosis was compatible with that of bullous emphysema. A catheter was then placed in the bulla and connected to a water-seal bottle (Fig. 1 *C*). Re-expansion of the left lung occurred in ten days with a resulting increase in vital capacity to 1,800 c.c. (Fig. 1 *D*). The same procedure was done on the right side (Fig. 1 *E* and *F*) where a similar cavity was found. The right lung re-expanded, except for a small apical portion, after three weeks of water-seal drainage, but the catheter became twisted and was removed. Another catheter was reinserted into the residual cavity at a later date. Continuous suction drainage by Stedman pump was necessary for an additional four weeks to obtain maximum re-expansion (Fig. 1 *G*). The patient's vital capacity was then increased to 3,550 c.c. Skin sensitization tests were all negative for extrinsic allergens. He is now capable of carrying on full daily activity and has had no complaints except for very occasional asthmatic attacks.



A. *B.*

Fig. 2 (Case 2).—*A*, Bronchogram showing depression of upper lobe bronchi from bilateral apical blebs. *B*, Postoperative x-ray film following decompression of blebs.

CASE 2.—G. R., a white man, 31 years of age, was admitted October, 1947, with complaints of gradually increasing dyspnea for the past four years, and pain in the right upper side of the chest for one month. He had been discharged from the Army in 1944 with a diagnosis of cystic disease of the left lung. X-ray examination revealed absent lung

markings in the upper two-thirds of the left side of the chest and the upper half of the right side of the chest. Bronchograms showed the compression of the upper lobe bronchi toward the bases of the lung (Fig. 2 *A*). Because of this depression it was possible to see several centimeters into both upper lobe bronchi at bronchoscopy. The usual three-stage Monaldi procedure was started on the left side. Thoracoscopy revealed a huge spherical cavity with thin, whitish translucent walls. An area 3 cm. in diameter at the base of the cavity showed some gray, shredded lung tissue with only a few visible bronchial communications. Because of this small area of denuded lung, it was felt that the diagnosis was compatible with that of emphysematous bleb. The bleb was decompressed, except for a small apical portion, within three weeks. The catheter then became twisted and had to be removed. It was found that the fenestrations in the tubing had been made too large and allowed the walls to collapse. An attempt was made to place a catheter in the residual portion of the bleb, but because of lack of pleural symphysis a surgical pneumothorax was obtained. After the lung had re-expanded a catheter was successfully placed in the bleb and it was quickly decompressed. The thoracoscopic findings in the right side of the chest were similar to those described on the left. Catheter suction drainage resulted in complete re-expansion of the right lung in two weeks (Fig. 2 *B*). The patient's vital capacity was increased to 3,350 c.c., and he was able to carry on vigorous activity without dyspnea.

CASE 3.—E. M., a 62-year-old white man, was admitted December, 1947, with complaints referable to chronic bronchitis and arthritis. The patient had had symptoms of chronic cough for ten years and gradually increasing dyspnea for the past two years. X-ray examination revealed absent lung markings in the upper one-third of the right side of the chest with some herniation of the mediastinum toward the left. Vital capacity was 1,370 c.c. Monaldi drainage was carried out with re-expansion of the right lung in four weeks. Biopsy revealed pleura and a few atrophic alveoli. The diagnosis was compatible with that of emphysematous bulla. Vital capacity was increased to 2,530 c.c.

CASE 4.—S. S., a 31-year-old Negro man, was admitted July, 1947, with symptoms of epigastric distress and pain in the left upper side of the chest. He was discharged from the Army with a diagnosis of chronic pneumothorax on the left. Complete gastrointestinal work-up was negative. Chest films revealed absence of lung markings in the upper one-third of the left side of the chest. Thoracoscopic examination showed bullous changes similar to those described previously. Decompression by electric suction pump was accomplished in the usual manner in three weeks. Vital capacity increased from 2,080 c.c. to 3,400 c.c.

CASE 5.—J. R., a 45-year-old white man, was admitted June, 1945, for treatment of peptic ulcer and chronic bronchitis. Ulcer symptoms were relieved with medical management. X-ray examination revealed two large emphysematous cavities in the left upper lobe with some evidence of old inactive tuberculosis. There was a small amount of fluid in the anterior cavity. Examinations for tuberculosis were all negative. The anterior cavity was decompressed with Monaldi drainage in three weeks. The fluid within the space proved to be sterile. Diagnosis from biopsy of the cavity wall was compatible with that of bulla. The patient refused further surgery and was discharged at this time.

CASE 6.—J. O. B., a 68-year-old white man, was admitted December, 1945, for treatment of severe dyspnea. He complained of asthmatic attacks and progressive dyspnea for twenty years. The patient was cyanotic even with maximum respiratory effort while sitting in a wheel chair. X-ray examination revealed absence of lung markings in the left side of the chest with the mediastinum shifted to the right. Bronchograms showed the terminal branches of the left bronchial tree compressed against the mediastinum (Fig. 3 *A*) and pushed anteriorly by a tension cavity located posteriorly (Fig. 3 *B*). Vital capacity was 800 c.c. After an extrapleural pack was inserted, the patient became more cyanotic and sank into coma. Because of the patient's extreme condition it was felt that decom-

A. *B.* *C.*

Fig. 3 (Case 6).—*A*, Bronchogram revealing absent lung marking on the left, compression of the bronchial tree, and shift of the mediastinum. *B*, Lateral film showing anterior compression of the bronchial tree and the posterior location of the emphysematous tension cavity. *C*, X-ray film during suction drainage demonstrating re-expansion of left lung.

pression was necessary at once. The gauze packing was removed one week after it had been placed in the extrapleural space, and a trocar and catheter were advanced into the cavity. The patient improved for one day and then showed signs of increasing dyspnea. X-ray pictures revealed the catheter in the bulla and also a tension pneumothorax. The pneumothorax was apparently due to a lack of pleural symphysis which allowed air to escape around the catheter into the pleural space. The pneumothorax was quickly decompressed by inserting another catheter. Re-expansion of the left lung was accomplished in one month (Fig. 3 *C*). When recently contacted, the patient was found to be active about his home.

CASE 7.—O. H., a 10-year-old white boy, was admitted for treatment because a routine chest film revealed a complete absence of lung markings in the left side of the chest, with a shift of the mediastinum to the right. The boy was completely asymptomatic and engaged in strenuous athletics. A three-stage drainage was done and a catheter placed into the cavity through the anterior part of the chest. Within a few days fluid, containing gram-negative organism, developed in the cavity and temperature went up to 104° F. Another large catheter was placed in the base of the cavity posteriorly. The fever mitigated and the cavity gradually reduced in size. At discharge, the left lung had re-expanded except for a small space in the left base containing a little fluid. The patient remains asymptomatic, and careful follow-up study is being carried out by periodic x-ray examinations. Because of the marked reaction following the insertion of the catheter it was believed that this may possibly have been an epithelial-lined cyst. This mistake could be avoided in the future by careful thoracoscopic study of the cavity and biopsy of the wall.

CASE 8.—W. M., a 52-year-old white man, had been hospitalized for over one year for incapacitating dyspnea. Asthmatic attacks began five years previously. Dyspnea had been progressive in the past two years. The patient was cyanotic and dyspneic at bed rest even when using all accessory muscles of respiration. There were findings of early right heart failure. Laboratory studies indicated a marked secondary polycythemia. The routine x-ray examination showed far-advanced emphysematous changes with areas of decreased density (Fig. 4 *A*). Bronchial markings could be seen extending to the periphery of the lung fields. However, the oblique (Fig. 4 *B*) and lateral films (Fig. 4 *C*) revealed definite bullous cavities. Catheters were inserted in the usual manner, into large bullae at both bases with decompression obtained in two weeks (Fig. 4 *D*). The patient has been relieved of some of the dyspnea and is semiambulant. Catheter drainage is contemplated for the remaining several large bullae. In advanced, multiple, bilateral bullae, there is a question of how long the patient will be benefited, and how often the procedure will have to be repeated. The fact remains however, that the patient is much improved at present.

CASE 9.—L. C., a 28-year-old white man, was admitted with symptoms of marked dyspnea on exertion. The patient was discharged from the Army in 1941 with a diagnosis of cystic disease of the right lung. He had had no symptoms referable to his chest up until that time. X-ray examination revealed only scattered markings in the right side of the chest and a definite displacement of the mediastinum (Fig. 5 *A*). After adhesions were produced, thoracoscopic examination revealed many thin-walled cavities that appeared like "soap-suds," which would rupture and coalesce when touched with the tip of the scope. A catheter was inserted (Fig. 5 *B*), but very little benefit was derived. Because of these findings an exploratory thoracotomy was done. Multiple cystic degeneration of the entire right lung was found and a pneumonectomy performed (Fig. 5 *C*). The patient states that his "wind" is now better than ever before in his life.

SUMMARY AND CONCLUSIONS

1. Patients with bullous emphysema are being seen by the thoracic surgeon with increasing frequency. In the past, the surgical treatment of emphysematous blebs and bullae has been considered hopeless.

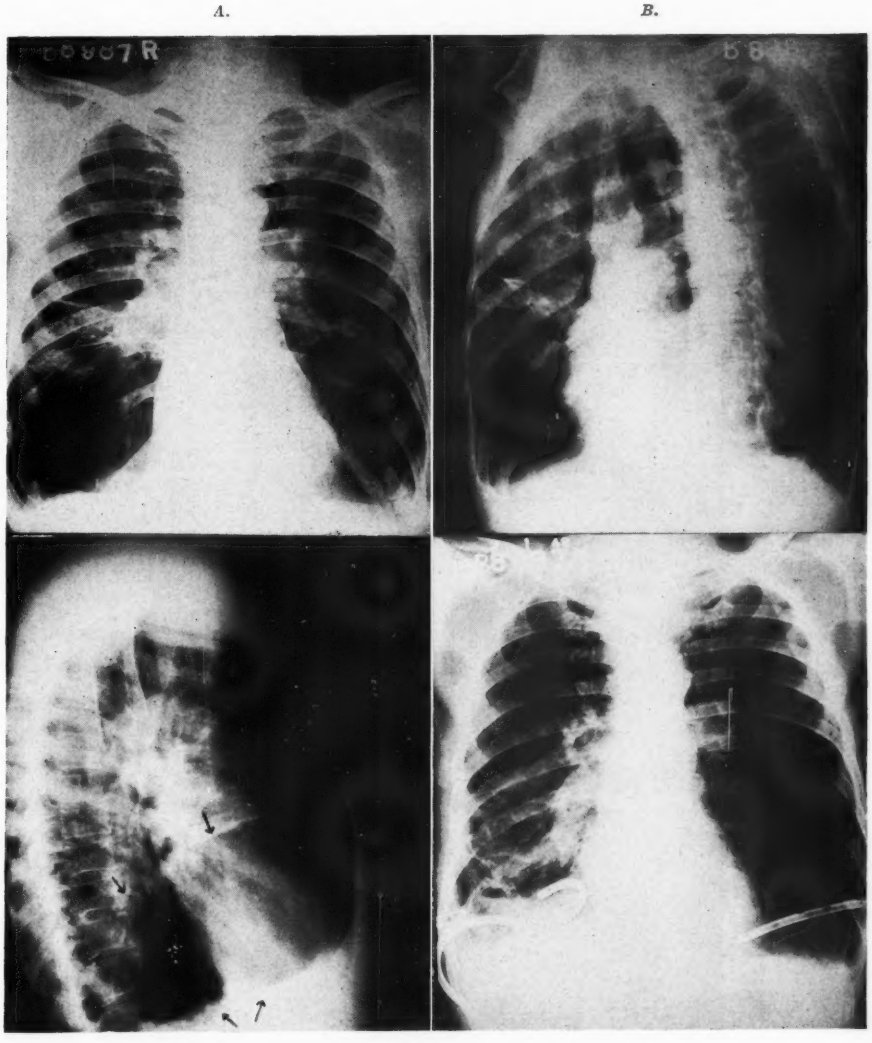
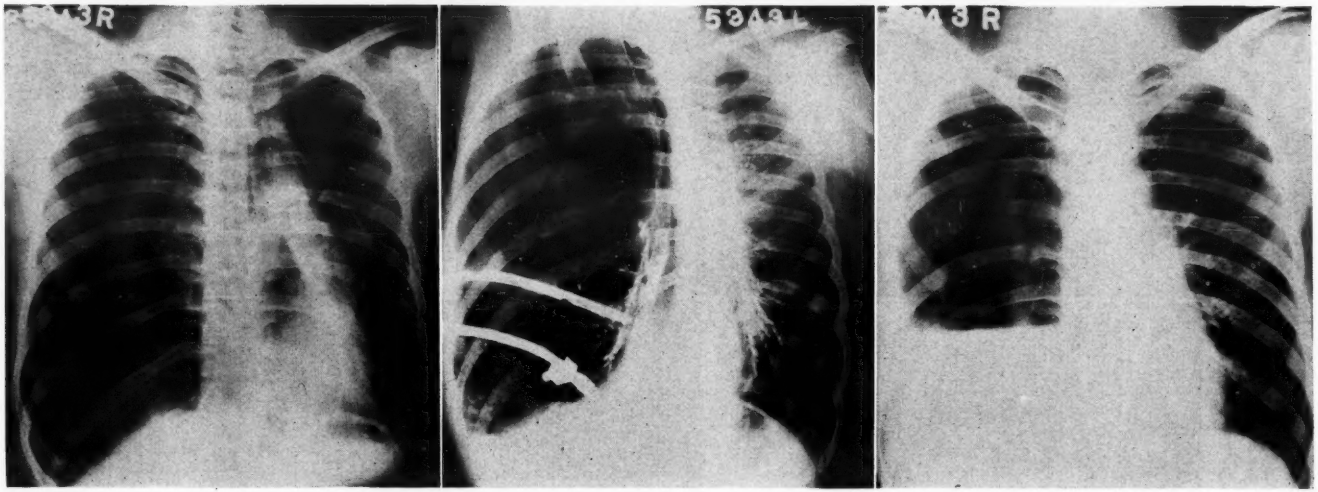


Fig. 4 (Case 8).—*A*, Bilateral severe emphysematous changes, with no definite cavities visible. *B*, Oblique film reveals absent lung markings at both bases. *C*, Right lateral x-ray film shows large bullous cavities. *D*, Catheters in bullae during decompression.



A. *B.* *C.*

Fig. 5 (Case 9).—*A*, Only scattered lung markings in right side of chest with mediastinal shift and pronounced herniation. *B*, Catheter in right cystic lung with very little re-expansion; note compression of right bronchial tree. *C*, Postoperative x-ray film following pneumonectomy. Mediastinum has returned to normal position and the compressed left lung has re-expanded.

2. The pathogenesis of tension cavities and their check valve mechanism is discussed. The x-ray and clinical picture is outlined.

3. The methods of treatment are reviewed and the intracavitary suction drainage procedure is presented. It is believed that this is the procedure of choice in certain types of emphysematous cavities. Eight cases are presented in which gratifying results have been obtained by this method.

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(See discussion on page 798.)

REVIEW OF SEVEN YEARS' EXPERIENCE WITH INTRACAVITARY (MONALDI) DRAINAGE OF TUBERCULOUS CAVITIES

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INTRODUCTION

IN DISCUSSING Dr. Head's[1] paper on intracavitary drainage before this Association in 1944, one of us (W. W.)[2] presented a summary of our results on 35 patients treated up to that time. Our series now represents 100 cavities in 90 patients who were accepted for intracavitary drainage between August, 1940, and Dec. 31, 1947. While treatment of 11 of these patients was begun during 1947, and thus their follow-up study has been limited, the remainder have been observed for one to seven years. The size of the group and the period of observation warrant this report.

In July, 1938, Monaldi[3] and his collaborators at the Carlo Forlanini Institute first described this method of treating tuberculous cavities. It consists in catheter drainage and the application of suction. While previously many surgeons had drained an occasional tuberculous cavity, the method was not extensively used until these modifications increased its simplicity and effectiveness. It is now a valuable adjunct to our surgical armamentarium in the treatment of certain tuberculous cavities. Since the original report, this method has been tried and studied in many countries, and Monaldi[4] stated that some 400 articles concerning it have now appeared in the world literature.[5-39] In support of this procedure, he has stressed certain theoretical considerations concerning cavities, the most important of these being that the size of a cavity may be out of all proportion to the amount of excavated lung. A tuberculous cavity arises from caseation of the pulmonary parenchyma with subsequent evacuation of the debris. A cavity enlarges by progressive evacuation of its caseous center and/or because of mechanical factors. These mechanical factors are both endocavitary and extracavitary.

The endocavitary pressure depends upon the patency of the draining bronchi. Many authorities feel that if a bronchus is completely blocked, air will be absorbed from the cavity and the walls will tend to approximate. More commonly, the affected bronchus is only partially closed by granulation or scarring and acts as a ball valve, making it possible for air to enter during inspiration but difficult to escape during expiration. In such a case, the intracavitary pressure becomes positive and the cavity tends to enlarge, become spherical, and may have a fluid level. This enlargement results in compression of the adjacent lung parenchyma. Such cavities have been variously termed pressure,[32] tension,[33] blocked,[34] or insufflation[35] cavities. When successive films show alteration in the size of the cavity without evidence of extension of dis-

Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

ease, the presumption is strong that the variation in size is due to changes in the intracavitary pressure.

The extracavitary factors which tend to keep a cavity open are those forces which exert traction from the outside. First, the normal lung is under constant tension to fill the larger hemithorax. Second, every inspiration by a normally functioning thoracic cage and diaphragm increases this tension. Third, when the mediastinum is distorted toward the opposite side because of disease, tension is increased. Fourth, scarring and fibrosis of lung parenchyma tend to increase tension on adjacent areas. All of these are extracavitary forces which tend toward cavity enlargement.

Theoretically, it would seem that the ideal case for drainage would be a large round cavity with or without a fluid level, with an adjacent area of compressed lung, and with little surrounding disease. In our experience this type has frequently responded dramatically to drainage, but we have succeeded in closing some in which there was no demonstrable positive pressure within the cavity. In other cases, although the cavity did not change appreciably in size, the patient experienced marked symptomatic relief from the decrease in cough and expectoration and the subsidence of toxic symptoms.

INDICATIONS

Although Monaldi[4] used this method as an independent procedure and stated that with it alone he had treated "approximately 100 patients who for some years have been back at work, enjoy perfect health and display no symptoms of illness," our indications have differed and have been similar to those presented to this Association by Head[1] and by Vineberg[1, 32] in 1944 and by Welles and Gordon[36] in 1947.

We have used intracavitary drainage primarily as a salvage procedure either for palliative relief in far-advanced bilateral cases or in the hope that by its use the patient might be prepared for more definitive surgery. A major indication has been the possibility of reducing the size of a large cavity preliminary to thoracoplasty in the hope that the thoracoplasty would need to be less extensive and would be more successful.

The extent to which it has been employed as a palliative procedure may be appreciated by considering the fact that all but one patient had contralateral involvement at some time. In most instances the contralateral disease was extensive. In 46 patients there was cavity, some form of collapse, fluid, or empyema on the contralateral side.

We have used it where there was acute tuberculosis or recent spread. Frequently it has been of little value in such cases, but there have been instances where the resultant improvement has rendered thoracoplasty or resection feasible.

It has also been used in certain patients in whom respiratory reserves were too limited to justify collapse or resection. In all questionable cases we have been guided by the advice of Dr. George Wright, who has undertaken the necessary pulmonary function studies in his laboratory of pulmonary physi-

ology at Trudeau Sanatorium. In two patients age was a relative indication, in that they were considered unsuitable for thoracoplasty.

Attempts were made to introduce catheters into residual postthoracoplasty cavities in 3 patients. Technically this has been difficult. In one the catheter could not be introduced on two attempts. The second was later converted to a cavernostomy. A third is too recent to evaluate. It is dangerous to insert a large trocar into a narrow slitlike cavity close to the mediastinum. One such attempt in 1948 was followed by fatal hemorrhage. Even if successful, one would expect only symptomatic improvement because of the rigidity of the regenerated thoracic cage. We feel, however, that this is an indication when resection or revision is not feasible.

Drainage is now employed as a preliminary to thoracoplasty for any large cavity, because such cavities are notoriously resistant to collapse by any type of thoracoplasty. The effectiveness of prethoracoplasty drainage in reducing the size of cavities has been observed by many surgeons.[32, 36] In our hands, when there has been a long interval between the anterior resection and the subsequent posterior thoracoplasty, the extent of thoracic collapse has been limited by the regeneration of the anterior rib ends. Our attempt to modify this will be described under Technique.

Table I presents an analysis of the indications in our series, and it will be seen that practically every case presented more than one indication.

TABLE I. INDICATIONS
(Frequently More Than One)

1. Bilateral pulmonary tuberculosis			89
Opposite side had:			
Cavity	27		
Unexpanded pneumothorax	8		
Empyema	4		
Thoracoplasty	6		
Paraffin plombage	1		
		46	
2. Large cavity*			51
3. Cavity with acute spread of disease			39
4. Limited respiratory reserve			11
5. Age of patient			2
6. Previous unsuccessful thoracoplasty			3
7. Refused thoracoplasty			1

*Most have had positive intracavitary pressures, but we have been unable to determine this fact except by manometric readings. X-ray interpretation has frequently been unreliable.

After the decision to perform intracavitary drainage has been reached, there are certain practical considerations, the most important of which are the size and location of the cavity. It is technically difficult to introduce a trocar into a small cavity. A large tension cavity in the upper lobe with little surrounding disease is the ideal indication. It allows the tube to be inserted anteriorly where it interferes the least with subsequent thoracoplasty. Lower lobe cavities should be approached posteriorly. This has many disadvantages. The chief one is that the location of the tube interferes with the normal thoracoplasty or resection incision; second, the motion of the scapula has resulted in dislodging the catheter on several occasions; and third, a catheter

in this position is frequently uncomfortable. Three patients having posterior drainage have had thoracoplasty and resection.

Aspiration of a cavity at the apex of the lower lobe through an anterior incision has been successfully accomplished in several instances, but we have abandoned it as we feel it carries an unjustifiable risk because it is impossible to be certain that the interlobar fissure is obliterated. Our two operative fatalities occurred in this group, the first from hemorrhage following the wounding of a branch of the pulmonary artery in the middle lobe, and the second from a pneumothorax due to traversing an unobliterated interlobar fissure. The subsequent empyema proved rapidly fatal.

For many of the patients subjected to intracavitary drainage, the only alternative would have been cavernostomy. Drainage is simpler and cleaner, the aftercare is less, and there is the advantage of continuous negative pressure in the cavity. In two cases the Monaldi was subsequently converted into a relatively small cavernostomy. The disease was arrested eventually in one patient but only after lobectomy in the other.

CONTRAINDICATIONS

Our only contraindications have been (1) a history of profuse hemorrhage, (2) a free pleural space, (3) a cavity too small to enter with a trocar or inaccessible to safe and simple drainage (such as near the hilum), and (4) multiple cavitation so extensive that even successful drainage of one or two cavities would offer no reasonable hope of palliative relief.

The greatest hazard in drainage is a free pleural space. The failure to find a pocket with the exploratory needle is not proof of pleural symphysis. A blunt trocar will frequently separate pleural surfaces when no space can be found by a needle.

TECHNIQUE

Technique of drainage is divided into two parts: the first is the obliteration of the pleural space, and the second is the actual insertion of the trocar. Various methods of obliterating the space have been advocated, including the intrapleural instillation of the patient's own blood, sulfanilamide crystals, or talc. We now resect the intercostal muscles, usually together with a section of the first and second ribs, sprinkle sulfanilamide crystals on the endothoracic fascia, and pack the wound with gauze, close the skin, and then remove the pack in five days. Table II gives the number of cavities for which each method was used. In 13 instances early in the series no demonstrable pleural space was found with the exploratory needle and the trocar was successfully

TABLE II. METHODS OF OBLITERATING PLEURAL SPACE

1. No demonstrable pleural space (exploratory needle)	13
2. Injection of blood	4
3. Pack without rib removal	18
4. Rib removal with or without pack	63

In 2 patients too ill for pre-Monaldi packing or rib resection, a pneumothorax space was found and there was no further attempt to insert the trocar.

introduced. There were 4 instances in which cavity closure was obtained with pre-Monaldi rib resection alone. Two followed anterior resections and 2 posterior resections.

The amount of rib removed depends upon anticipated later treatment. If it is a purely palliative procedure with no hope of later thoracoplasty, extensive lengths of the upper two or three ribs are taken; however, the effect of extensive decostalization on pulmonary function must be considered. If thoracoplasty is deemed likely, we remove the first costal cartilage and a reasonable length of the first rib. If it is necessary to remove a bit of the second rib, this is taken lateral to the costal cartilage and as small a section as possible removed. This is a recent change in technique and has been necessitated by the relatively poor ultimate collapse if the anterior segments have regenerated before the thoracoplasty is performed. If a small segment of rib and costal cartilage has been left anteriorly, it is possible, at the time of the thoracoplasty, to remove the remaining anterior segments through a small separate anterior incision. This permits a better collapse. At the time of posterior thoracoplasty, rib resection is always terminated short of the point at which we are likely to break into the sinus tract. On two occasions the sinus tract was inadvertently entered. In neither instance did infection develop.

Insertion of the catheter under fluoroscopic control is advised. When possible, this has been performed two to three weeks after the packing. The location and depth of the cavity are ascertained with a 19 gauge aspirating needle, and when the cavity is entered, manometric readings are obtained. From Table III we see that in 47 cavities aspirated the pressures were positive; in 26, they were neutral; and in 9, they were negative.

TABLE III. MEAN CAVITY PRESSURE

Positive (plus 1½ to plus 18)	47
Neutral	26
Negative (minus 2½ to minus 6)	9

Occasionally, sputum will be obtained through the aspirating needle. When this occurs, it is proof that the needle is in the cavity. In one instance the cavity disappeared when air was withdrawn, and it was impossible to insert the trocar. This cavity subsequently reopened and was closed by thoracoplasty without further attempt at Monaldi drainage. There have been instances in which it was impossible to locate the cavity with a needle. In one such instance we were forced to conclude that the film had been misinterpreted and the cavity, if present, was much smaller than it seemed.

The Kupka modification of the Monaldi trocar is used. The safety flange is set at the proper depth, as determined by the aspirating needle. The needle is withdrawn and the trocar is inserted at the same point, in the same direction, and to the same depth. Manometric readings are again verified; the catheter

(No. 13 French) is inserted as far as it will go and the trocar withdrawn. The catheter is secured to the skin by a clove hitch of heavy silk fastened with adhesive. This holds the catheter without compressing the lumen. The position of the catheter is immediately checked by stereoscopic and lateral films.

Complications of insertion have been minor. The most common has been bleeding. This was of sufficient extent in 20 per cent to be noted on the record. In certain instances the bleeding had vitiated pressure readings or rendered it wise to discontinue the attempt temporarily. One hemorrhage, which was later fatal, occurred when the trocar was inserted despite the fact that brisk bleeding was encountered with the aspirating needle. There was one air embolism with temporary hemiplegia. Small pneumothoraces have on several occasions been a complication. If one is discovered with the aspirating needle, further attempt should be delayed. Pneumothoraces which are not found until after the insertion of the catheter are more troublesome. In only one instance was it necessary to aspirate air. One developed an empyema which proved fatal, as previously mentioned.

Table IV summarizes the complications of trocar insertions. Of the 100 cavities originally accepted for drainage, there was no attempt at trocar insertion in 9; in 7 of these the pre-Monaldi procedure resulted in such improvement that drainage was considered unnecessary, and in 2 others the attempt was not made because of the rapid deterioration in the patient's condition.

TABLE IV. TOTAL TROCAR INSERTIONS—113 IN 91 CAVITIES*

COMPLICATIONS DURING INSERTION		33 (29%)
1. Bleeding (1 led to fatality)		23 (20%)
2. Air embolus (nonfatal, temporary hemiplegia)		1
3. Pneumothorax pocket (1 led to fatality)		9 (8%)
(a) Pack without rib removal	2	
(b) No pre-Monaldi	3	
(c) Rib removal, no pack, but talc and sulfonamides	1	
(d) Rib removal, no pack	1	
(e) Present method	2	
(1) Resultant pyopneumothorax from traversing unobliterated fissure, and death (Case 81)		
(2) Pneumothorax without complications, tube not inserted		

*More than one attempt to introduce trocar:
2 patients had 4 attempts
3 patients had 3 attempts
11 patients had 2 attempts

POSTOPERATIVE CARE OF MONALDI DRAINAGE

Suction is not applied for twenty-four hours. Thereafter, a constant negative pressure of –20 to –30 cm. of water is maintained for twenty-four hours a day. For this purpose one of us (W.W.)[37] has devised a cabinet with a small electric motor, a constantly registering aneroid manometer, and a mercury safety valve which can be set for any level. Early in the series,

pressures as low as –50 and –100 were used temporarily while the secretion was thick and tenacious, but it was felt that such highly negative pressures were not safe, as it was observed that the amount of sanguineous drainage was increased. If there is frank bleeding, suction is discontinued for one or two days. In two patients the occurrence of large hemorrhages made it advisable to abandon the procedure and withdraw the catheter. Monaldi[4-7] felt that an intermittent negative pressure is better than a constant one, in that it involves less risk of traumatizing the tissue. We prefer to maintain a constant negative level.

It is not uncommon for the sinus tract to become secondarily infected, and the amount of secretion may become profuse. Occasionally a chest wall abscess has developed around the catheter. This has been incised and drained. Streptomycin, when used either intramuscularly or by topical application, has cleared many of them promptly. Certain patients, however, have lost resistance to the tubercle bacillus to such an extent that the adjacent soft tissues have melted away and the disease has progressed rapidly. Otherwise, dressings are not a problem. Catheters are usually replaced monthly. At the time of the first change the catheter is usually shortened so that its tip barely reaches the posterior wall.

Blood or blood-tinged drainage was recorded in 45 per cent. It was usually a minor problem and cleared when suction was discontinued for a few days. In only two instances was it so profuse that the catheter was withdrawn and the method discontinued. Table V summarizes the late complications of the series.

TABLE V. COMPLICATIONS AFTER INSERTION

1. Bleeding through catheter (2 severe enough to force withdrawal of catheter)	37 (45%)
2. Chest wall abscess or sinus around tube	13 (16%)
3. Perforation of posterior cavity wall with resultant empyema; thoracoplasty and thoracotomy	1

EFFECT OF CAVITY DRAINAGE

As a general rule there was marked clinical improvement almost immediately. Some patients have a mild febrile course for the first few days, but thereafter most of them experienced a sense of well-being with the reduction in the size of the cavity. Probably the most significant factor in this improvement was the external drainage of the sputum, with the resultant decrease in cough and expectoration. With the relief of symptoms there tended to be an increase in appetite and a gain in weight. DesAutels and Hayes[38] published detailed studies on several of the early patients in this series upon whom follow-up study had been done up to one year.

Cavities tend to close rapidly when subjected to drainage. Table VI is an analysis of cavity behavior. Of the 82 into which a catheter was introduced, 40 were eventually closed without further therapy, 33, within six months. In 33 other instances the cavities became smaller, and in only 9 did they remain

TABLE VI. EFFECT OF MONALDI ON CAVITY DRAINED

1. Cavity closure		
Within 1 month	6	
1 to 3 months	21	
3 to 6 months	6	
6 months or more	7	
		40 (49%)
2. Cavity smaller		33
3. Cavity unchanged or larger		9
Total drained		82

unchanged or become larger. In Case 2 the cavity could not be visualized at twenty-four hours.

Tubercle bacilli may disappear from the drainage in one month or less. In the presence of positive sputum and negative drainage another focus is suspected. An interesting observation has been that occasionally drainage has become negative while a cavity is still present. Table VII gives statistics on the disappearance of tubercle bacilli from sputum and drainage.

TABLE VII. SPUTUM AND DRAINAGE STATISTICS
(82 Cavities in 76 Patients)

	DRAINAGE (CAVITIES)	SPUTUM (PATIENTS)
1. Time of initial disappearance of tubercle bacilli by concentrated smear:		
1 month	5	9
2 months	15	6
3 months	6	5
4 months	6	4
5 months	2	1
6 months or more	6	9
	40*	34
2. Always positive	42	42
Total	82	76

*Of the 40 negative drainages the sputum remained positive in 18.

Extensive bacteriologic and pathologic studies have been performed by others on tuberculous cavities under various conditions. Steenken, D'Esopo, and Wolinsky[39] have reported their studies on excretion of streptomycin into tuberculous cavities. In 24 samples of intracavitary drainage in patients who were receiving streptomycin intramuscularly in dosages of 1.8 to 2 Gm. daily, they found an average concentration of 7.7 μg. per cubic centimeter, varying between 1 and 18 μg. The average concentration of the drug in the blood at the same time was 13.3.

RESULTS OF TREATMENT AND LATER PROCEDURES

One hundred cavities in 90 patients were originally accepted for treatment. In 7 instances the preliminary rib resection was followed by dramatic improvement. In 4 of these the cavities closed and in the other 3 they became so much smaller that drainage was thought unwise and thoracoplasty was performed in one. In 9 patients, repeated attempts were made to introduce the trocar, but were unsuccessful because of technical factors. These factors were

usually the size or location of the cavity. Two patients were so ill that, following the preliminary stage, it was deemed unwise to proceed further. Catheters were actually inserted in 82 cavities in 76 patients. Four patients had 2 cavities drained and 1 patient had 3. Table VIII summarizes the entire group.

TABLE VIII. ACCEPTED FOR MONALDI

	PATIENTS (90)	CAVITIES (100)
1. Pre-Monaldi resulted in improvement so Monaldi not done (cavity closure in 4)		7
2. Catheter could not be introduced		9
3. Catheter not introduced because of deterioration in patient's condition		2
Total drainage	76*	82
Sex: 53 males, 37 females Age: Average, 33 (range from 19 to 61 years)		

*Four patients had 2 cavities drained, 1 patient had 3 cavities drained.

Forty cavities were closed by drainage and, of these, the catheters were removed or extruded from 7, in 6 patients, and the cavities have remained closed for periods ranging from eight months to four and one-half years (Table IX). In eleven others (Table X), the cavities reopened in from three

TABLE IX. CATHETER OUT—CAVITY REMAINED CLOSED
7 Cavities in 6 Patients

TIME ELAPSED SINCE REMOVAL OF CATHETER	NUMBER
8 months	1
3 years	3
4½ years (1 bilateral)	3

weeks to fifty-nine months. Obviously there is no assurance that a cavity may not reopen long after the removal of the catheter. In certain instances in which this has occurred, it has been possible to re-establish drainage. It is logical to perform thoracoplasty, when possible, rather than to trust that the cavity will remain closed.

TABLE X. TUBE OUT AND CAVITY REOPENED IN 11 PATIENTS

INTERVAL BETWEEN REMOVAL OF CATHETER AND REOPENING OF CAVITY	NUMBER
Less than 3 months	2
3 months to 1 year	7
26 months	1
59 months	1
Total	11

When should a catheter be removed? We do not know. We have seen no ill effects from maintaining drainage indefinitely. One patient, Case 2, has had drainage continuously since August, 1940, because she refuses to allow the catheter to be removed, despite the fact that she has had a negative sputum and has been working full time in a sanatorium for several years. In general, in those patients for whom drainage is a palliative procedure and for whom

no other therapy is available, we have left the catheters in indefinitely unless they were extruded.

For those patients who have been sufficiently improved by drainage to justify collapse or resection, we have not withdrawn the catheter until definitive surgery has been completed and, if possible, until the resected ribs have regenerated. Following catheter removal, most sinuses healed promptly, although streptomycin has occasionally been necessary. There have been instances of subsequent reopening after initial healing. This has usually been associated with reopening of the cavity.

The most heartening aspect of this method has been the number of patients who have been successfully prepared for further surgery. It has been noted that as one cavity becomes smaller, an adjacent cavity may become larger. For that reason we have felt that, when a cavity has been controlled by drainage, the improvement should be secured by thoracoplasty, if possible. Table XI shows that 30 patients have already had definitive surgery, and there is an excellent prospect that ultimately it may be possible for an additional 6. Thus 33 per cent of the entire group have already had the benefits of subsequent therapy. In only 76 patients, however, was drainage successfully carried out. There were 5 others* who obtained sufficient improvement from the pre-Monaldi so that it was thought wise to omit tube insertion. Of this total of 81 patients, 30, or 37 per cent, have already received more definitive therapy. In addition, there are 6 others who may yet have the advantage conferred by permanent collapse.

TABLE XI. SUBSEQUENT PROCEDURES

Thoracoplasty	25	
Cavernostomy	1	
Cavernostomy and lobectomy	1	
Thoracoplasty and pneumonectomy or Pneumonectomy and thoracoplasty	3	
Total who had more definitive procedure	30	(33% of entire series)
Thoracoplasty probable	6	
Total who have had or may have more definitive procedure	36	

Some of the thoracoplasties have not been as mechanically perfect as we should wish because of the regeneration of the anterior stumps before the posterior collapse could be completed, and also because, in view of limited respiratory reserves, we have not dared to make the collapse as extensive as the amount of the disease warranted.

Table XII is a summary of the present status of the 90 patients. Only 19 are regarded as "apparently well" and 31 are already dead. We anticipate that, with time, several of the remaining 40 will be classified as "apparently well." It should be remembered, too, that 11 of these patients were accepted

*As seen in Table VIII, there were 7 patients with improvement following the pre-Monaldi procedure. In this group 2 patients had 2 cavities each, 1 drained, 1 not drained. Both of these 2 patients have already been tabulated in the total of 76 patients who had drainage. Accordingly, only 5 can be counted in the present tabulation.

TABLE XII. PRESENT STATUS

Apparently well:		
Monaldi alone	7	
With subsequent procedure	12	
		19
Still curing:		
Cavity closed	3	
Subsequent procedures	14	
Tube still in	12	
(To have thoracoplasty, 6)		
Tube out, cavity open	6	
Tube never inserted	5	
		40
Dead:		
After subsequent procedures	6	
All others	25	
		31
Total		90

for treatment in 1947 and there has not been a chance for the disease to be arrested.

While the mortality rate is already high, it is encouraging that so far 19 patients have apparently been reclaimed by this method. Many others who have not had arrest of their disease have had their lives prolonged with marked amelioration of their symptoms.

Of the 31 deaths, only 2 can be attributed to the treatment per se.

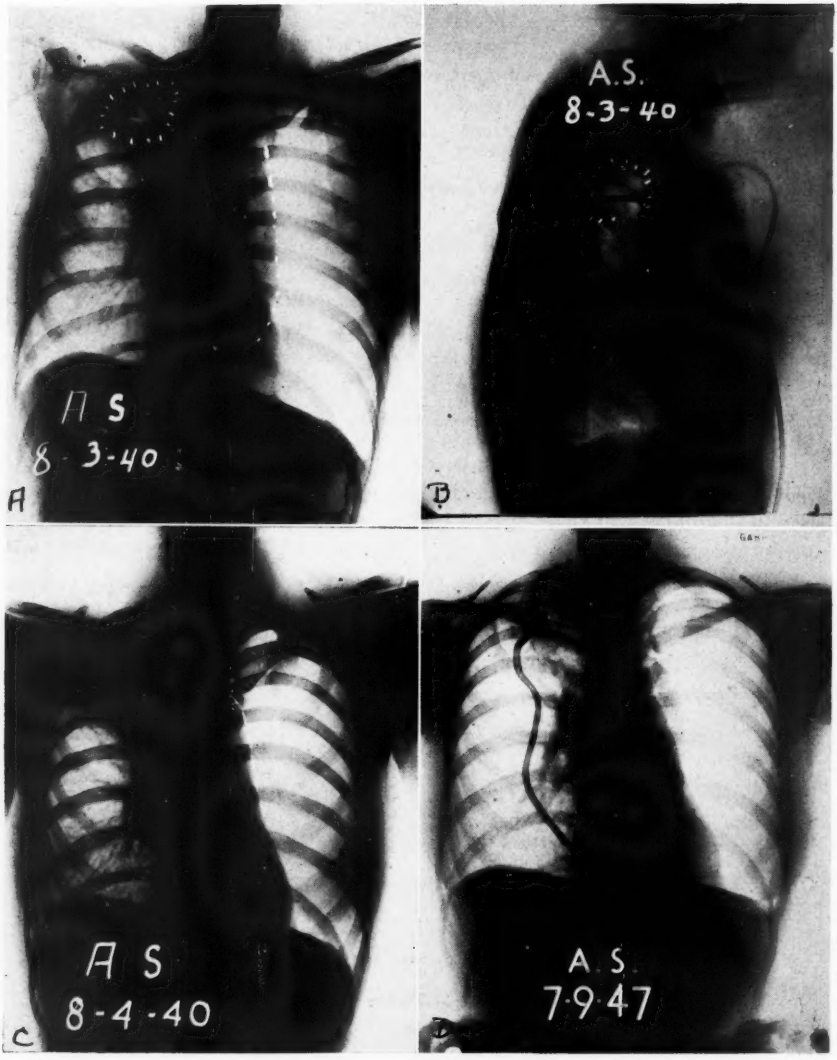
Tables XIII and XIV show the time and cause of death in all cases.

TABLE XIII. DEATHS AS RELATED TO TREATMENT

1. Tube never inserted		5
2. Early, within 1 month		
Hemorrhage	1	
Empyema	1	
		2
3. Late		
Less than 1 year	11	
1 to 2 years	6	
2 to 3 years	2	
3 to 4 years	3	
Over 4 years	2	
		24
Total		31 (34.4%)

TABLE XIV. CAUSES OF DEATH

Progression of disease	21
Postthoracoplasty, postpneumonectomy, 1	
Withstood thoracoplasty, 2	
Hemorrhage, either side	5
Empyema	1
Air embolism, late	1
Withstood thoracoplasty	
Pulmonary insufficiency	1
Postthoracoplasty	
Nontuberculous	2
Hypertension and nephritis, 1	
Suicide, postthoracoplasty, 1	
Total	31



Group I: Four cases are presented in which Monaldi alone was used (Cases 2, 18, 33, and 67).

Fig. 1 (Case 2).—A. S., woman, aged 27 years; indication: palliative. *A*, Bilateral disease, onset, 1929, left; pneumothorax, left, February, 1936; unexpandable lung, bronchial stenosis left main, tuberculous; cavity, right, note fluid level; sputum 60 c.c., Gaffky VII; temperature to 99.2° F. Pleural space, right, found to be obliterated. Respiratory function studies on July 5, 1940, revealed extremely low maximum minute ventilation. Catheter inserted Aug. 3, 1940. *B*, Lateral view showing posterior location of cavity, with catheter in place. *C*, Twenty-four hours later, cavity not visualized. *D*, Present status: Cavity not visualized since Aug. 4, 1940; sputum and drainage negative since 1941; working full time as medical secretary for several years. Patient refuses to have catheter removed, but we believe it should have been removed several years ago. She still applies suction at night. (Note: Clinical progress of this patient during the first year of drainage was published by DesAutels and Hayes.[36])

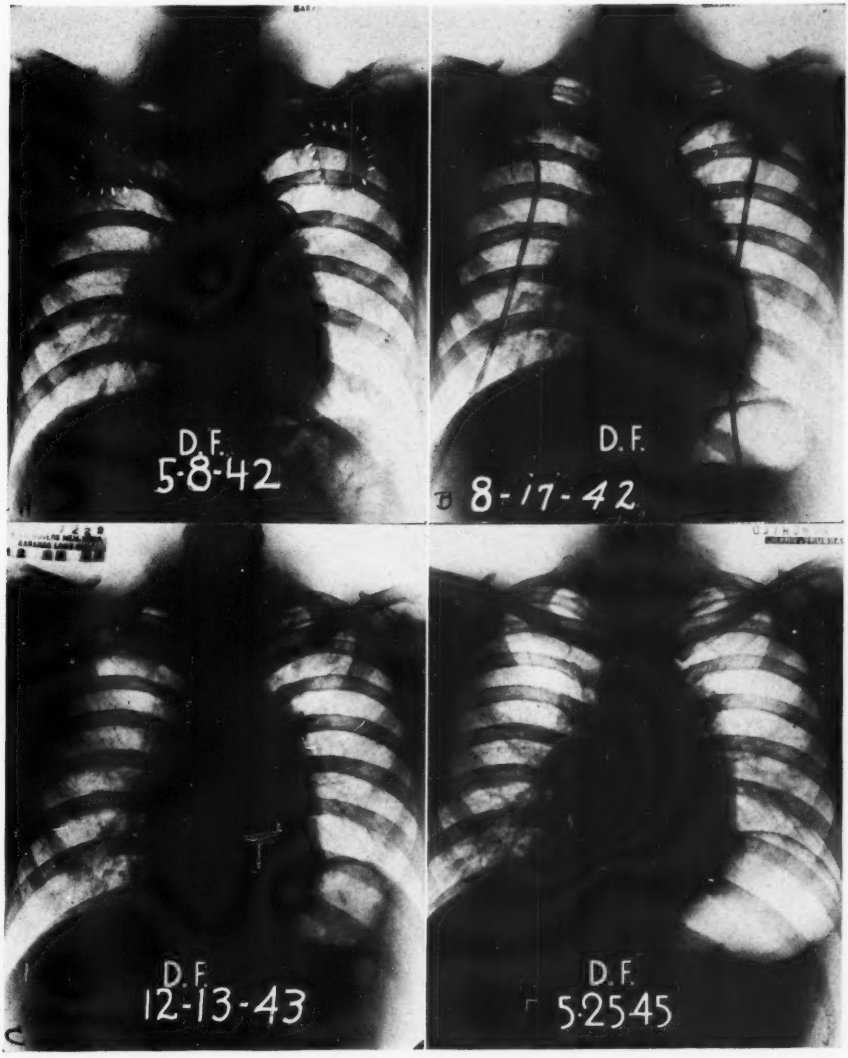


Fig. 2 (Case 18).—D. F., man aged 21 years; indication: palliative. *A*, Bilateral apical cavities; 25 c.c. Gaffky IV sputum; temperature 97 to 99° F.; bilateral pre-Monaldi packing, first interspace, without rib resection, Jan. 29, 1942; catheter inserted, right, May 8, 1942. *B*, Right cavity not visualized, drainage negative, June, 1942; catheter inserted, left, July 14, 1942; marked immediate decrease in size of cavity; possible development of new cavity medially. *C*, Drainage from both catheters negative after October, 1942; catheter, right, removed June, 1943; cavity left, not visualized after October, 1942; catheter, left, removed September, 1943. Phrenic, left, Nov. 3, 1943, for small adjacent cavity. *D*, Repeat phrenic, left, Jan. 5, 1945; patient discharged; both cavities have remained closed; sputum negative since Dec. 13, 1943. Letter from patient, March 31, 1948, stated "working on farm, feeling fine, have not had an x-ray for some time."

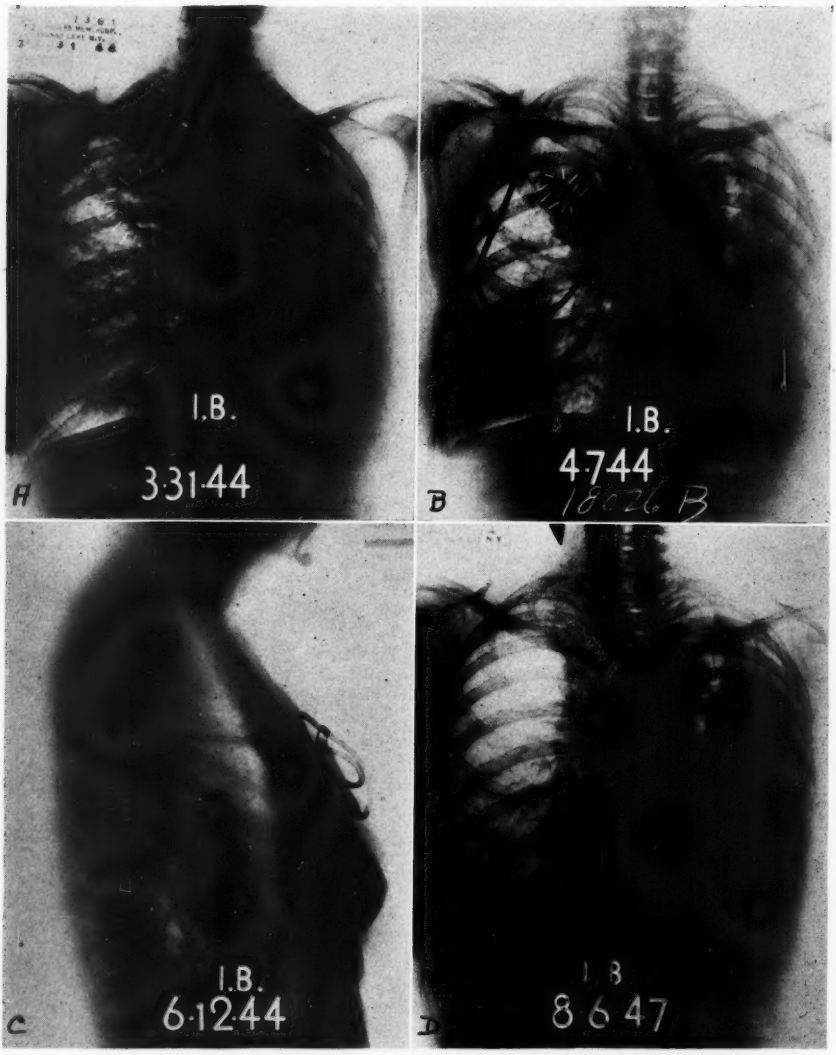


Fig. 3 (Case 33).—I. B., woman, aged 47 years; indication: palliative. *A*, Bilateral disease; pneumothorax, left, 1933 to 1938; lung failed to re-expand, pleural cavity filled with bloody serous fluid; sputum Gaffky VIII; temperature to 99.4° F.; cavity, right; pre-Monaldi rib resection, anterior end of first rib, Feb. 10, 1944. *B*, Catheter inserted, April 7, 1944; pressures +12, −6; first drainage Gaffky X. *C*, Lateral view; position strongly suggests cavity in apex of lower lobe; if true, catheter traversed fissure without incident. *D*, Cavity not visualized since June, 1944; drainage negative, July and August, positive Oct. 5, 1944; sputum negative Oct. 5, 1944; drainage negative Oct. 16, 1944; catheter removed November, 1944; present status: cavity closed, sputum negative; has remained well, doing own housework.

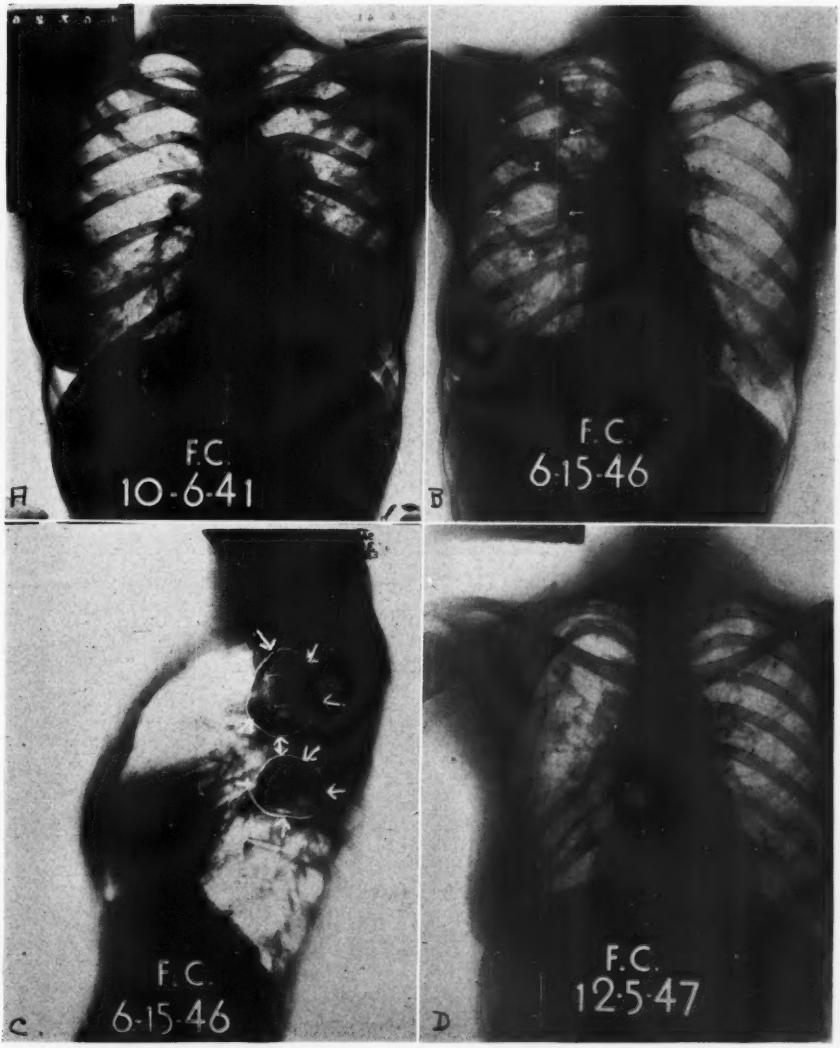
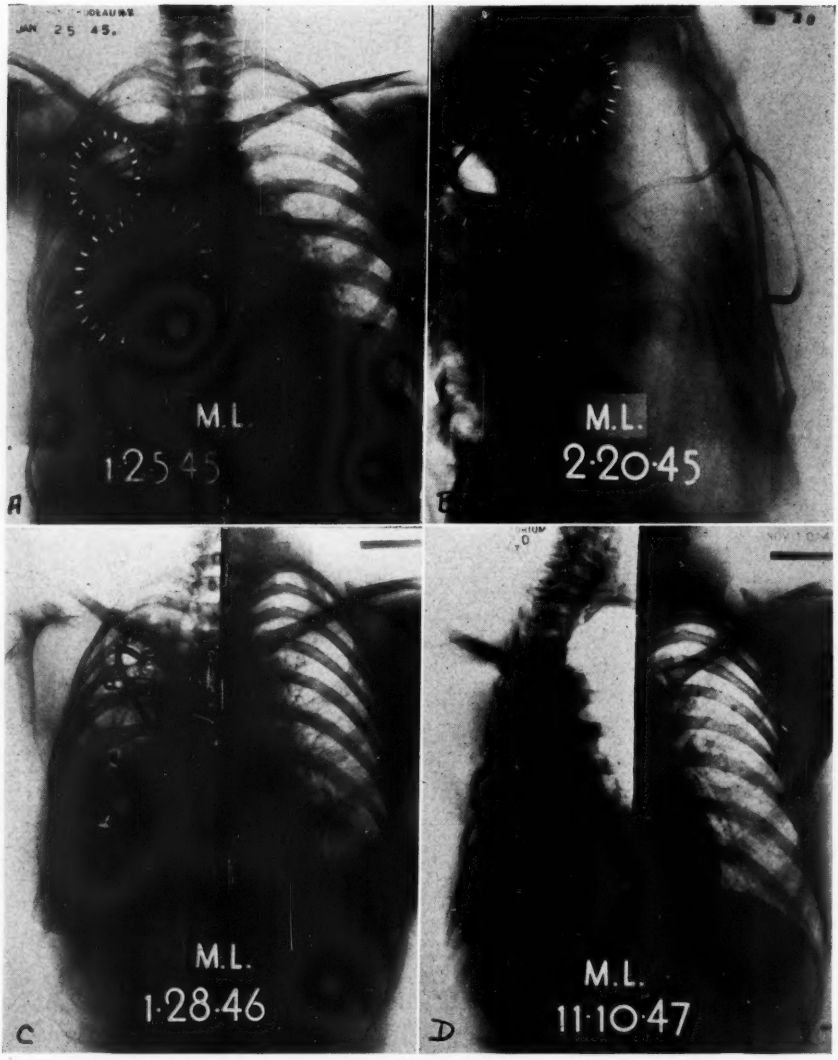


Fig. 4 (Case 67).—F. C., woman, aged 21 years; indication: palliative, pulmonary function not adequate for thoracoplasty or resection. *A*, Early film, at age of 16, five years before entering this series; extensive infiltration, left, with involvement of right. *B*, Had pneumothorax, left, for 8 months, 1941 to 1942; phreniclasis, left, Nov. 5, 1943; discharged from sanatorium Dec. 7, 1944; readmitted, February, 1946, with development of two large cavities, right; pneumothorax, right, instituted April 8, 1946, but abandoned because of increase in size of cavities. Note residual pneumothorax pocket and fluid levels in both cavities. Pneumonectomy considered but respiratory function studies, June 8, 1946, by Dr. George Wright, showed maximum minute ventilation of 70 liters and bronchospirometry revealed 0_2 absorption 48 per cent on the right and 52 per cent on the left. Despite the cavities on the right, that side was carrying approximately the same respiratory load as the left because of extensive damage to the left by the disease of five years previously; patient, therefore, not suitable for either resection or thoracoplasty, right. *C*, Lateral film showing posterior location. This also shows why this lung is still functionally important. *D*, Pre-Monaldi resection the first to third anterior rib ends, on June 20, 1946; posterior pre-Monaldi resection of the fifth to eighth ribs on Aug. 6, 1946; disappearance of lower cavity by film of Aug. 26, 1946; catheter inserted into upper cavity, right, through anterior approach, Aug. 28, 1946; cavity apparently closed; catheter removed April 16, 1947. Patient in sanatorium Dec. 31, 1947, asymptomatic, with no cavity by planigrams, and sputum negative since January, 1947.



Group II: These two cases had drainage and thoracoplasty (Cases 45 and 66).

Fig. 5 (Case 45).—M. L., woman, aged 32 years; indication: palliative, possible prethoracoplasty. *A*, Acute bilateral disease; febrile; toxic; marked weight loss; giant cavity formation, right upper and lower. *B*, Resection of anterior ends of the first to fourth ribs on Jan. 20, 1945; insertion of two Monaldi catheters, right, Feb. 14, 1945; lateral view, note that lower catheter has traversed the oblique fissure without incident. *C*, Prethoracoplasty film showing marked reduction in size of cavities; patient's condition much improved. *D*, Thoracoplasty completed March 30, 1946; upper Monaldi catheter removed April, 1946; lower catheter removed Oct. 14, 1946; present status (nineteen months postthoracoplasty) markedly improved, small amount of positive sputum, residual basal cavity by planigram.

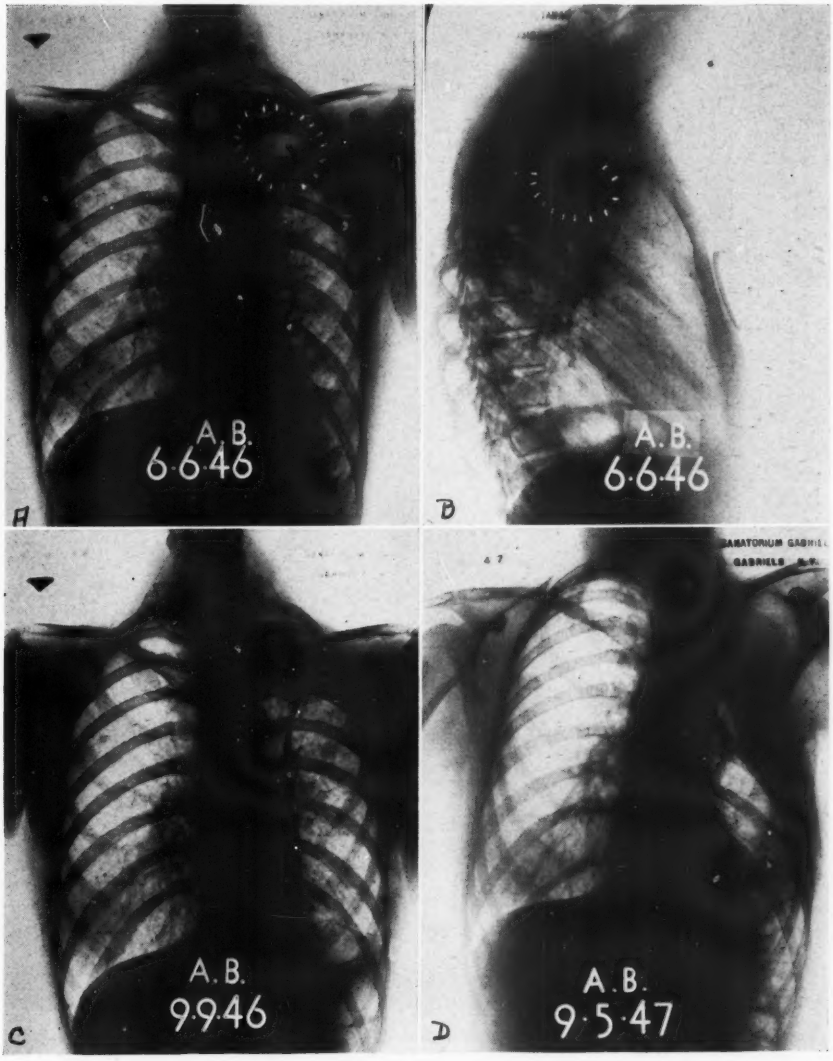
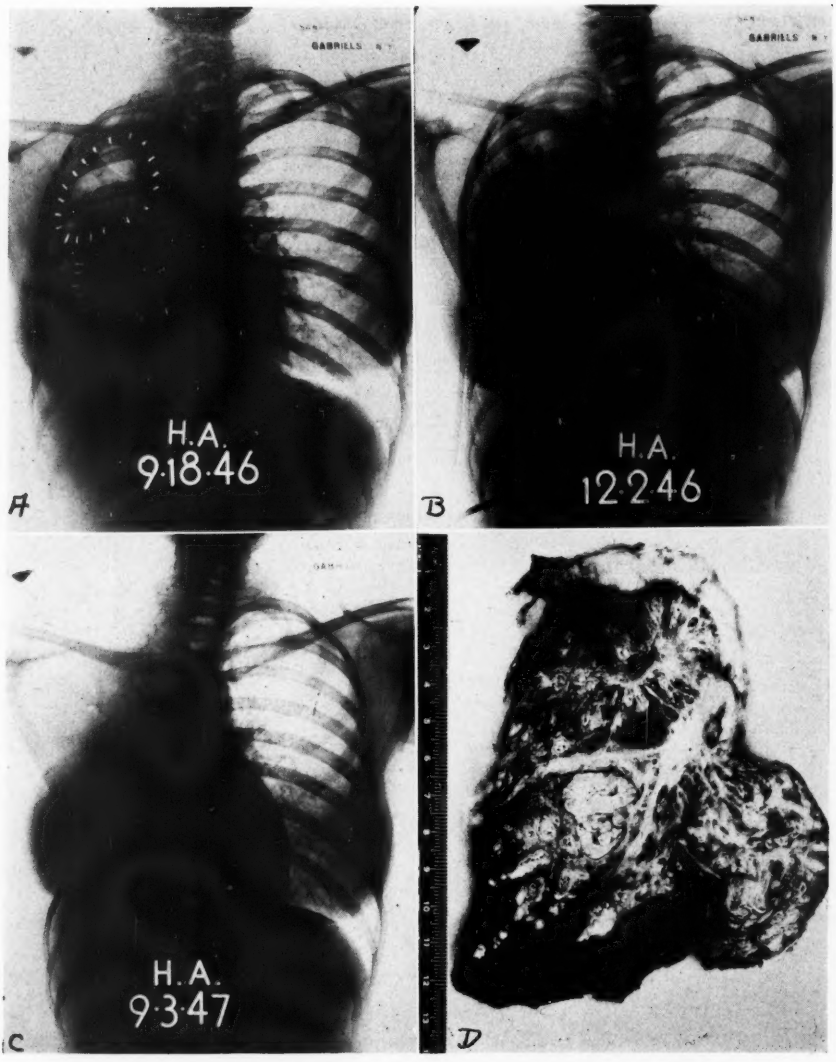
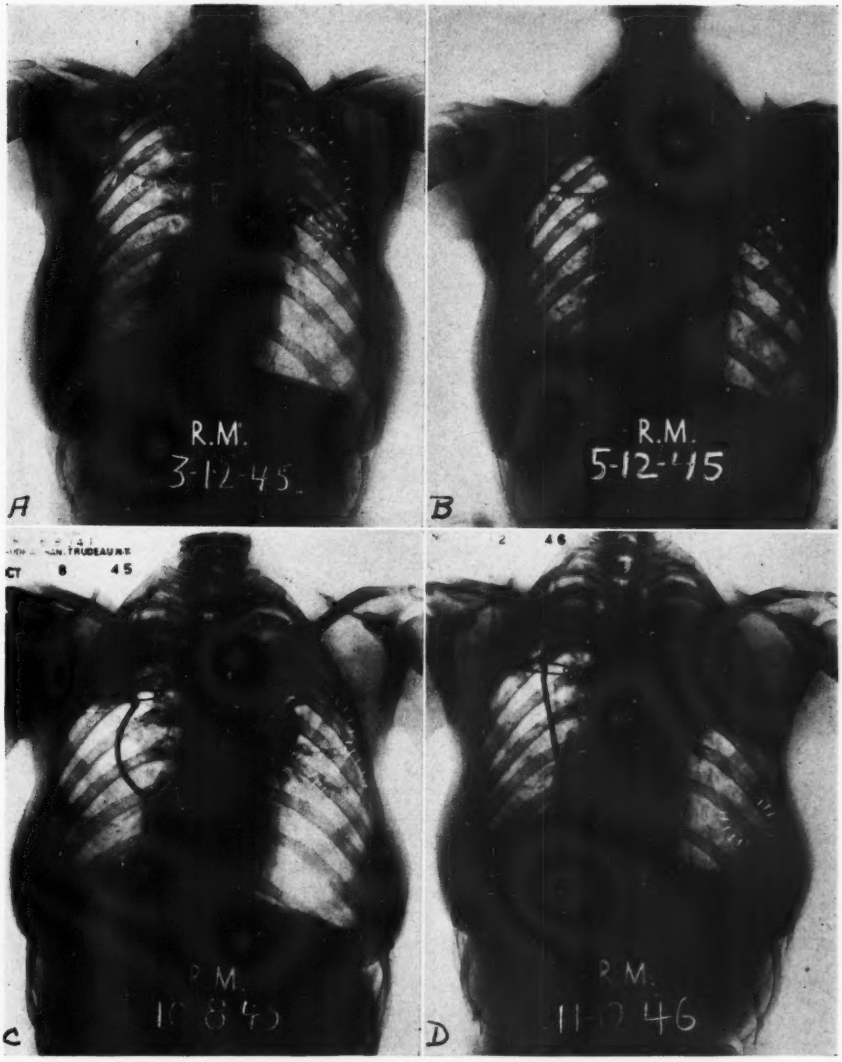


Fig. 6 (Case 66).—A. B., woman, aged 35 years; indication: palliative, possible prethoracoplasty. *A*, Bilateral tuberculosis with recent spreads; cavity, left, with fluid level; pre-Monaldi resection of anterior ends of the first to third ribs on May 16, 1946; catheter inserted June 6, 1946. *B*, Lateral view. *C*, Cavity closed; sputum and drainage negative. *D*, Seven-rib thoracoplasty completed, December, 1946; cavity closed and sputum negative as of Dec. 31, 1947.



Group III: One patient had Monaldi followed by resection and then thoracoplasty.

Fig. 7 (Case 71).—H. A., woman, aged 23 years; indication: palliative, possible prethoracoplasty. *A*, Acute disease, right, with two large cavities; patient raising 100 Gm. of Gaffky V sputum; temperature to 100° F. *B*, Posterior pre-Monaldi performed Sept. 19, 1946; with resection of segments of the seventh to tenth ribs; two posterior catheters inserted Oct. 16, 1946; marked reduction in size of cavities, symptomatic improvement, sputum Gaffky II, temperature not above 98.6° F. *C*, Lower catheter removed, January, 1947, cavity apparently closed at that time; pneumonectomy, right, through anterior incision, Feb. 27, 1947, and remaining catheter removed; thoracoplasty completed May 24, 1947. Present status: sputum negative; small residual draining sinus; insidious increase of disease, left. *D*, Section through resected lung. Note contracted scarred lung. Cavity in upper lobe not visualized; caseous focus in mid-lung apparently remnant of lower cavity.



Group IV: Two patients were followed from initial drainage to death (Cases 52 and 81).

Fig. 8 (Case 52).—R. M., woman, aged 31 years; indication: palliative. *A*, Bilateral disease, extensive cavity formation both lungs. In 1932 large cavities, right and left; artificial pneumothorax, right, 1932 to 1934; artificial pneumothorax, left, 1936 to 1941; began working, 1941; June, 1944, hemoptysis; development of cavity on right; later, two cavities, left; at present, sputum Gaffky IX, temperature to 100° F. *B*, Pre-Monaldi, bilateral, March 29, 1945; left, no free pleural space; right, anterior section of first rib removed; catheter inserted in left upper cavity on April 27, 1945, pressures +5, −2. *C*, Insertion of catheter on right, June 29, 1945, pressures +3, −3; drainage both sides, Gaffky II; no appreciable change in size of cavities. *D*, Insertion of second catheter on left, May 16, 1946; through large sinus, left anterior part of chest wall; bilateral improvement of cavity-bearing areas that were drained; development of new cavity (fourth) left base; sputum and drainage positive; death, Feb. 12, 1947, progressive tuberculosis.

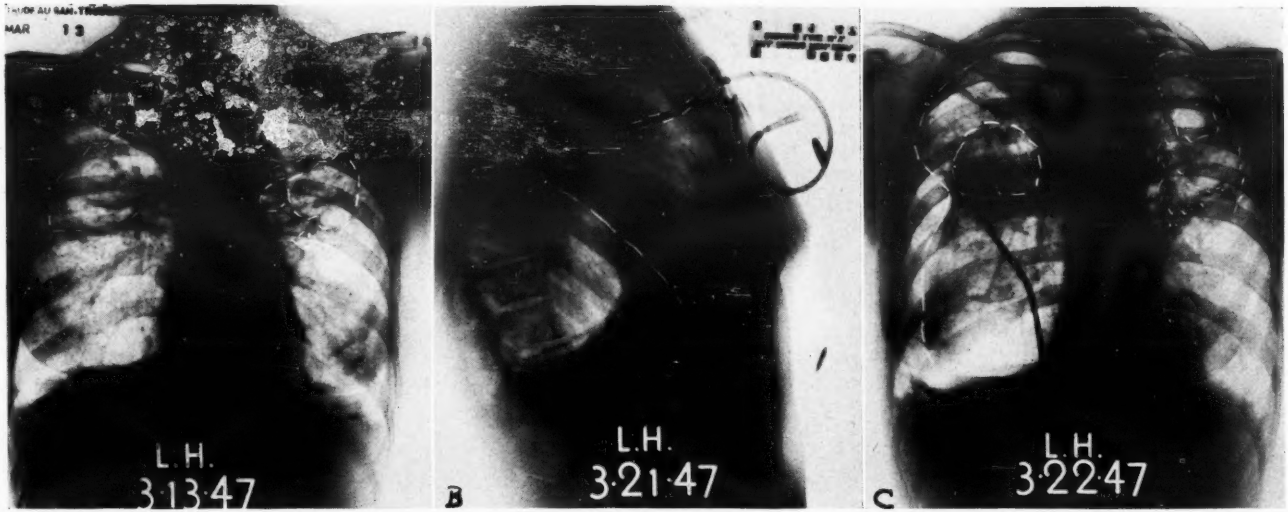


Fig. 9 (Case 81).—L. H., woman, aged 30 years: indication: palliative. *A*, Bilateral cavitary disease; excessive cough and expectoration. *B*, Lateral film, anterior rib resection Jan. 14, 1947, catheter inserted anteriorly. March 21, 1947, traversing oblique fissure. *C*, Pneumothorax resulting from trocar traversing oblique fissure. Patient developed fulminating pyopneumothorax; death on March 28, 1947. Autopsy revealed symphysis of pleurae at site of preliminary packing but no obliteration of the interlobar fissure. This represents an error in judgment. There was almost no possibility of controlling all of the cavities and the hazard of traversing a fissure was unjustified.

Figs. 1 to 9 show, by a series of films and accompanying legends, the progress of 9 representative patients who have been subjected to intracavitary drainage.

SUMMARY

1. Intracavitary drainage has been used as a salvage procedure, both as a palliative in otherwise hopeless cases and as a method of preparing certain patients for collapse therapy. Without this help most of them could not have had their final surgery.

2. The method has been described, detailed tables presented, and several representative series of films shown.

The cooperation of the following physicians and sanatoriums has made this study possible: Dr. John N. Hayes, Dr. J. Woods Price, Dr. Henry W. Leetch, Dr. Francis B. Trudeau, Dr. Daniel M. Brumfiel, Sanatorium Gabriels, Will Rogers Memorial Hospital, Trudeau Sanatorium, Northwoods Sanatorium, and Broadacres Sanatorium of Utica.

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DISCUSSION ON "INTRACAVITARY SUCTION (MONALDI) IN THE TREATMENT OF EMPHYSEMATOUS BULLAE AND BLEBS" BY DR. JEROME R. HEAD AND DR. EDWARD E. AVERY,* AND "REVIEW OF SEVEN YEARS' EXPERIENCE WITH INTRACAVITY (MONALDI) DRAINAGE OF TUBERCULOUS CAVITIES" BY DR. WARRINER WOODRUFF, DR. WINFIELD O. KELLY, AND DR. ALLAN STRANAHAN

DR. PAUL C. SAMSON, Oakland.—The differentiation between so-called blebs and intrapleural pulmonary cysts is a very important one. There is too much confusion in the literature between these two entirely different pathologic entities. I would question, however, whether one should split hairs, as it were, in trying to differentiate blebs and bullae; it seems to me that the pathology is so similar that there is little reason for trying to distinguish between them.

In so far as treatment is concerned, the authors have agreed that Monaldi drainage is, for them, the treatment of choice. I cannot argue too strenuously with that because I have never tried the Monaldi procedure, but I can with some assurance speak of the surgical treatment of the lesions, and I believe that our results are good enough for surgery to be considered not infrequently in treating these conditions.

(Slides.) In the first place, the pathology is such that I am unable to tell how Monaldi drainage can completely relieve the condition. This slide shows multiple cystic blebs laid open in the upper lobe. They were not visualized in the x-ray examination. There are

*See page 761 for article by Head and Avery.

interlacing strands of alveolar septa and multiple fistulas in the base. These are not tension cysts; when the lung is removed from the body they all collapse.

Dr. Dugan and I have performed operations on 7 individuals; one was a bilateral lobectomy and is the only case in which total lobectomy was done.

In the other patients, excisional surgery was performed by clamping across normal lung beneath the base of the cyst, excising the cyst and the subjacent battered parenchyma. There was minimal sacrifice of normal lung. In one patient, 5 cysts were removed, varying in diameter from 8 cm. to 3 cm. Such an experience speaks for a surgical, rather than a suctional approach in many instances. We have had immediate re-expansion of the lung in all cases. Of course, many of these patients may appear to be in poor physical condition prior to operation, because of diminished vital capacity. This situation is remedied immediately, however, as soon as the thorax is open and the giant bleb deflated.

DR. W. E. BURNETT, Philadelphia.—I would like to commend Dr. Head and Dr. Avery on this very ingenious way of approaching these most difficult problems. I wish I had known about this sooner, but in the past we have used the surgical approach, as Dr. Samson has done. It is noteworthy that in these patients, as has been called to our attention this morning, many times the cysts are not demonstrable; I am using the term cyst, meaning blebs and bullae. A great many have not shown identifiable allergy, although a good many have been improved by bronchial relaxants and aerosol of penicillin. One patient that seemed too diffuse for operation has greatly improved clinically. Dr. Rosemond and I have operated on approximately 11 of these patients, with great relief up to three years in all but 2.

(Slides.) As you see, this patient had an extremely large cyst. It was resected, and it was very simple to remove this large cyst without removing any significant amount of lung. In another patient, we have taken out several cysts from the right side, including parts of each lobe, without doing a lobectomy; we reserve that for intrapulmonary cysts. As long as the lesion can be recognized, it certainly seems wise to remove it, despite the fact that there is decrease in pulmonary function. After operation the function rapidly improves. The compressed lung of one man, who had had a similar large cyst in the chest for five years, was seen to re-expand rapidly at operation when the bulla was removed and positive pressure was applied by the anesthetist.

We have comparative studies on the more recent cases, and postoperative studies on even the earlier cases. One man had a reserve of 43 pr cent before operation, 23 liters maximum breathing capacity, with 79 per cent oxygen utilization on the right and 21 per cent on the left. Postoperatively, the 43 per cent reserve was increased to 70 per cent, with the distribution equal between the two lungs. We think this is an effective method of dealing with large blebs or bullae, but the lesion must be localized, not diffuse, and extremely ill patients may present too much risk. The simpler, although more prolonged, method used by Dr. Head and Dr. Avery offers great value, particularly since the disease is usually a progressive one, and therefore, any surgery which does not remove the cause is only palliative, although a great deal of relief can be afforded in properly chosen cases. At any rate, great effort should be made to find and eradicate etiological factors in combination with either method.

DR. ARTHUR VINEBERG, Montreal.—The subject of large tension cavities should be reviewed from a different point of view. The procedure of in tracavitary drainage is a useful one. It should be employed as a preliminary procedure in the treatment of all tension cavities. The practice of treating moribund, or nearly moribund, cases by intracavitary drainage throws discredit on the procedure. The so-called salvage cases do badly no matter how they are treated.

In reviewing a group of thoracoplasty results in Montreal, we have not been pleased with the number of persistently positive cases. The thoracoplasties have been performed by many different surgeons in our district; usually the collapse appears to be excellent.

Most of these cases, however, showed residual cavitation. We believe these were probably tension cavities at the time of thoracoplasty and should have been treated by anterior stage thoracoplasty, followed by intracavitary drainage.

The indications and contraindications for intracavitary drainage of tension cavities should be the same as for thoracoplasty. The application of this procedure to salvage cases should be discontinued.

Dr. Woodruff reported that intracavitary drainage had caused hemorrhages which resulted in the deaths of a number of his patients. This is to be expected if a metal trocar is introduced blindly into the lung.

I would like to mention a technique of intracavitary drainage first reported by us in 1942, which we believe to be reasonably safe. Briefly, the cavity is first entered by a fine needle, which acts as a guide for cannulae to be threaded over it in series into the cavity. By this simple technique it is possible to introduce a soft rubber catheter into a pulmonary cavity with a minimum of danger. We believe that an anterior stage thoracoplasty done prior to intracavitary drainage helps to close the cavity. When it is impossible to proceed to posterior stage thoracoplasty, the residual fistulous tract is opened and packed in order to close the bronchial opening and prevent reinflation of the cavity. We avoid packing of cavities.

I should like briefly to mention our results in the treatment of 43 cases with giant tension cavities. We have divided the cases into those with unilateral disease only and those with bilateral involvement. In the unilateral group of 21 cases, there were 15 patients alive and well, or approximately 71 per cent. Of the 22 cases with bilateral disease 68 per cent are dead.

DR. RICHARD MEADE, Grand Rapids.—I have had the opportunity of seeing many of these patients reported upon by Dr. Head and Dr. Avery, and have been impressed, as you have been, with the success of the method. I should like to add one point. In certain cases when there is spontaneous pneumothorax due to rupture of the cyst, the failure of re-expansion is due to the fact the lung has become encased in an inelastic membrane. No suction will help that, and open operation is the answer. Even if one is not able to remove the cyst, it is surprising how the lung will expand after decortication and suction can then be applied until the lung becomes adherent to the chest wall.

DR. EVARTS GRAHAM, St. Louis.—I think this suggestion of Dr. Head and Dr. Avery is one of great importance. Over a period of years I have handled these patients and have had my "fingers burned" many times. I have resected these cysts; I have done lobectomies; I have plicated the cyst. I have had splendid results in some cases, and in others the patient has returned with a large bullous cyst somewhere else in the lung.

These patients are often desperate risks for surgical procedures. I have been much intrigued, therefore, by this ingenious method recommended to us. Although it may take time for the lung to expand, time means nothing to these patients who are so handicapped. One question is, is it going to be permanent? Is the cyst going to be permanently collapsed and become so adherent that a new cyst cannot form? It seems to me possible that this procedure might be supplemented by insufflation of powder through a catheter, which would result in forming adhesions between the walls and the cyst, and thereby prevent its reforming.

DR. A. L. BROWN, San Francisco.—Some years ago we presented here the Eloesser flap for drainage of these cysts, and the answer to the question of whether these cysts might recur, I think, might be that flap; I think it is of particular value there. I believe it is well worth while to induce reduction in the size of the cyst with continuous suction like the Monaldi, and when it is reduced, to prevent re-expansion of the cyst, it is worth while to introduce a skin flap which remains open as long as there is a fistula which permits decompression to the outside. This tends to aid in closure, and should the cyst re-expand years later the little dimple that is left gives an immediate point of entrance.

DR. DAVID H. WATERMAN, Knoxville.—I am sure we all agree with Dr. Woodruff that Monaldi drainage has a definite place in tuberculosis therapy. I, also, am inclined to believe that this group of patients under discussion should not be called salvage cases any more, in that we now have a way to take care of them.

Our own experience is similar to that of Dr. Welles, reported before this Society at the last meeting, in the use of Monaldi drainage in the so-called giant cavity, whether tension or not, in conjunction with thoracoplasty. Often thoracoplasty alone fails to achieve a good result. With the use of the Monaldi in addition we have so far not failed to secure cavity closure.

I should like to bring out one point in surgical technique. I think it is agreed that preliminary anterior thoracoplasty before the Monaldi tube is inserted is of value, in that the subsequently infected drainage tract need not be transversed in completing the thoracoplasty. In performing the anterior stage, we make separate small incisions over the two ribs to be removed, and resect the rib segments without disturbing the intercostal muscles between them. Oxycel gauze is placed along the periosteal beds to stimulate pleural adherence. A few days later the Monaldi catheter can be inserted safely through the intercostal muscle bundle. Within a few more days the first posterior stage of thoracoplasty can be performed. In this way the waiting period is much reduced between the anterior and the posterior thoracoplasty stages.

DR. CAMERON HAIGHT, Ann Arbor.—It is important to evaluate the late results of a procedure such as Monaldi drainage, which was initially greeted with so much enthusiasm. Dr. Byron Evans and Dr. Mahelz Molins have analyzed the results in 31 patients operated upon in our clinic during the five-year period beginning on Jan. 1, 1940. The results have not been as encouraging as had been hoped, and they have been less favorable than those reported by Dr. Woodruff, Dr. Kelley, and Dr. Stranahan. In order to evaluate the results, the cases have been divided into four groups according to the indication for the operation.

The first group consisted of those cases in which thoracoplasty alone was not expected to close the cavity. Following an initial anterior thoracoplasty, the Monaldi drainage was done prior to the posterior thoracoplasty. The results in this group were encouraging; 8 of the 12 patients had closed cavities, although the sinus track remained open in 2. There was 1 residual open cavity, and there were 3 deaths in this group. The second group consisted of only 2 cases in which Monaldi drainage was used after completion of the thoracoplasty. In 1 case the cavity was closed, but the sinus track was open and cultures of sputum or of drainage material from the track were intermittently positive. The other case in this group was untraced. The third group consisted of 10 poor-risk patients for whom Monaldi drainage was done only in the hope that they would become suitable for thoracoplasty. The results in this group were discouraging, although 1 patient had an unexpected closure of the cavity from the drainage alone and 2 were improved. Of the remaining 7 cases, 5 died and 2 were untraced. The fourth group consisted of 7 desperately ill patients for whom Monaldi drainage was done for palliation only. Two of the patients had bilateral operations. All of the patients in this group died, either as a result of progression of the disease or as a result of complications of the drainage operation.

The complications of Monaldi drainage have included varying degrees of bleeding in 11 patients, it being fatal in one. A spread of the tuberculosis occurred in 5 of the patients following an initial anterior thoracoplasty. Two patients developed meningitis secondary to the drainage, and 1 developed empyema.

We feel that Monaldi drainage may play a large part in obtaining a successful result by thoracoplasty in patients having inflation cavities, especially huge ones, with highly efficient bronchial check valves, making thoracoplasty alone unlikely to succeed. In the other indications we have been disappointed with the results of the procedure, but we have not used it as an independent measure for solitary cavities in early unilateral tuberculosis, as recommended by Monaldi, because we believe the results are better from phrenic paralysis,

pneumothorax, thoracoplasty, and other collapse therapy procedures. Our restriction of Monaldi drainage to patients presenting unsuitable indications for other forms of treatment when used alone probably accounts for our relatively poor results, as compared with others' series, in patients with a better prognosis.

DR. J. D. MURPHY, Oteen.—I wish to make two brief points. In a series of 15 patients we have used Oxycel gauze* to cause a fusion of the pleurae. In fusion of the pleurae previous to the Monaldi, a site is selected nearest the peripheral point of the cavity, and under general anesthesia an intercostal incision is made down to the endothoracic fasical plane. The parietal pleura is dissected with finger and gauze dissection over an area sufficiently large to insert a 4 by 4 square of Oxycel. The wound is then tightly closed. After ten days to two weeks we have found the pleurae fused and have inserted a catheter through a trocar in the usual manner. We have occasionally resected a piece of rib in making this dissection. This modification saves the patient considerable time in that one stage of operation is avoided, that of removal of a gauze pack and subsequent delay of insertion of the catheter.

These 15 patients have been a part of a series of attempted salvage cases. The patients have had extensive bilateral disease with a large cavity in one apex. The bilateral disease was so advanced as to preclude consideration for major surgery. Following the insertion of the Monaldi catheter and initiation of pneumoperitoneum therapy, there has been a distinct improvement in this group and we have already performed a thoracoplasty with conversion of sputum in 5 of them. The thoracoplasty was preceded by an average of six months' pneumoperitoneum therapy with catheter in place.

DR. EDWIN J. GRACE, Brooklyn.—The big problem of the tension cavity I do not think can be solved by using detergent antibiotic therapy as an aerosol. In fact, this patient became progressively worse by the inhalation method, and it was found unsatisfactory. (Slides.) As you can see the tension cavity became enormously enlarged as a result of using the aerosol method. While waiting to produce a chemical obliterating pleuritis over the posterior aspect of the tension cavity, in order to put the detergent antibiotic synergistic solvent directly into the cavity, the patient left Brooklyn to see Dr. Overholt, who performed a lobectomy. A recent follow-up study eight months after the operation provided us with the information that the patient is doing very well.

DR. HEAD.—My participation in this work has been very happy. I suggested that it might work, and Dr. Avery has made it work. He has been responsible for taking care of these patients and for the surgery done upon them.

Dr. Samson said that it was not important to distinguish between bullae, blebs, and cysts. I feel that it is important. Progress in medicine and surgery depends very much upon increasingly fine distinctions between disease entities. Lack of discrimination and loose and inexact use of terms always create confusion. Bullae, blebs, and cysts are distinct and different conditions, differing in etiology and pathology and in the indications for treatment. It is very important that we do differentiate between them. In doing so it is interesting to remember that it was William Snow Miller, to whom we are all indebted in so many ways, who described the important etiological and anatomical differences between bullae and blebs.

Dr. Graham has asked whether, once deflated, these blebs remain so, or whether, as in the case with tuberculous cavities, they re-open. So far all of them have remained closed.

DR. WOODRUFF.—I just want to comment on the discussion of Dr. Vineberg. I think this procedure definitely has a place in the treatment of large tension cavities. We are no longer performing thoracoplasties over large cavities without previous decompression. I should like to show two slides which illustrate why we have modified our prethoracoplasty procedure.

*Manufactured by Parke, Davis & Company, Detroit, Mich.

(Slide.) This slide shows the condition before Monaldi drainage: There is a cavity on one side and a partially unexpanded lung on the other. Moderately long segments of the first and second ribs were removed before the drainage. When the thoracoplasty was performed seven months later, there had been so much regeneration of the anterior stumps that it was impossible to get a complete collapse. (Slide.) This slide shows the thoracoplasty which, although adequate in this instance, is technically poor due to the long regenerated struts of the anterior rib ends.

Because of several similar results, we have modified our technique in those instances in which we anticipate subsequent thoracoplasty. We now take a fair length of the first rib and its cartilage but a very short segment of the second rib and no cartilage. Following the first or second posterior stage of the thoracoplasty, we then remove the second cartilage. This procedure aids in mobilization of the anterior chest wall, and its use has resulted in better collapse.

COARCTATION OF THE AORTA

Report of Six Cases With Operation

H. Brodie Stephens, M.D., and Orville F. Grimes, M.D. (by invitation)
San Francisco, Calif.

COARCTATION of the aorta is not a rare congenital anomaly. It occurred as the primary lesion in 85 of Abbot's 1,000 cases of congenital heart disease, and as a complicating anomaly in 93 others. Two anatomic types of coaretation, infantile and adult, are recognized. The infantile type is usually accompanied by other more severe congenital defects and in most instances death occurs in infancy. It is characterized by a narrowing of the aorta between the origin of the left subclavian artery and the insertion of the ductus arteriosus. This condition normally exists in the fetus, where the ductus arteriosus carries blood into the descending aorta while the aorta proximal to the ductus is narrowed. In the so-called adult type, usually seen in adults and in children past infancy, there is a sharp constriction or obliteration of the descending aorta at or near the site of insertion of the ductus. The most popular explanation of the etiology of this type postulates that the obliterating action of the ductus, which occurs soon after birth, affects the neighboring portion of the aorta. This interpretation leaves unexplained the fact that in almost 10 per cent of cases of coaretation the ductus is patent.

Hypoplasia of the aorta is a frequent complicating congenital defect. The condition of the aorta proximal to the site of coaretation is now of more than theoretical importance in view of the development of a surgical attack on this defect. While the proximal aorta is hypoplastic in about 10 per cent of cases, in at least 50 per cent there is dilatation of the proximal aorta. This is probably due to the increased pressure in that segment and possibly to an inherent weakness of the aortic media. When the proximal aorta is greatly dilated and weak, rupture may occur. This catastrophe occurred in 38 of Abbot's cases.

Coaretation definitely shortens the life of the individual, although symptoms may not appear until the patient is well into adulthood. Cardiac decompensation is the leading cause of death and once this appears, the patient's course is speedily downhill. Spontaneous rupture of the aorta, cerebral accidents, and mycotic endarteritis are also frequent causes of death.

For reasons that are only partially known, in patients with coaretation the vascular system proximal to the coaretation is subjected to various stresses which render these tissues liable to inflammatory or degenerative changes. This is of great significance, not only as an added indication for early surgical intervention, but also because it indicates the difficulties attendant upon such intervention. Aortic tissue that has been subjected to hypertension for decades is not the ideal tissue to hold sutures while being pounded by the blood stream.

From the Department of Thoracic Surgery, University of California Medical School.

Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

Consequently these patients should be operated upon early in life before irreversible changes in the vascular system have developed.

The central theme of this paper is a review of the case histories and operations of six patients in whom coarctation of the aorta was verified.* It is our hope that the presentation of this group of cases will provide informative data to a subject still young in the annals of operative endeavor.

CASE REPORTS

CASE 1.—W. S., a 16-year-old boy, entered the University of California Hospital Oct. 12, 1947. After a bout of bronchopneumonia at the age of 7 year, he had recurrent attacks of asthma occasionally requiring medical aid. He complained of bilateral calf pain, noted most often while at rest following exercise. Suboccipital, supraorbital, and bitemporal headaches usually followed severe exercise so that he had long since learned to limit his activity. He was aware of coldness in the lower half of his body while the upper torso and arms might be perspiring from the effects of environmental temperature. There was no dyspnea, chest pain, palpitation, cyanosis, or edema of the lower extremities.

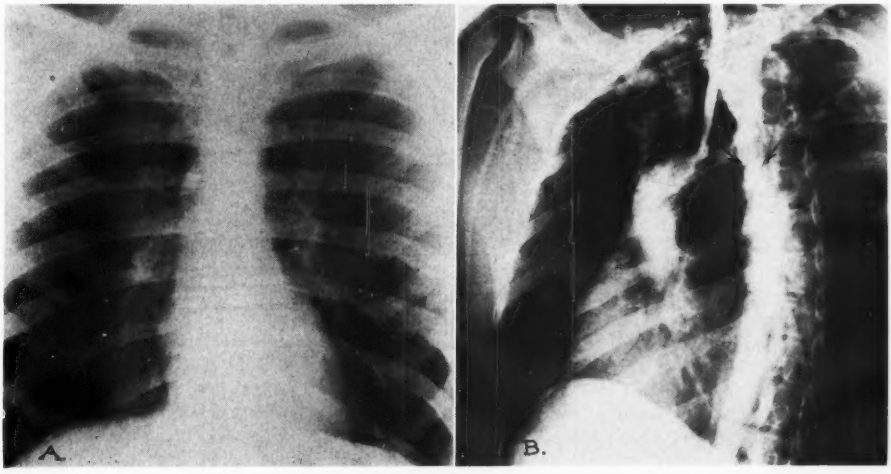

Fig. 1 (Case 1).—The heart was not enlarged and its contours were normal (*A*). Retrograde carotid arteriography demonstrated that the transverse aortic arch was hypoplastic. The coarctation (*arrow*) was located immediately distal to the point of origin of the left subclavian artery. The latter was at least twice normal size (*B*).

The heart was not enlarged and its rhythm was regular. A systolic murmur was heard posteriorly at the level of the third to fifth ribs in the paravertebral line. The arterial pulsations in the upper extremities were normal in quality. The peripheral tension in the right arm was 180/105, and in the left 160/105 mm. Hg. The abdominal aortic and femoral pulsations were barely discernible and those below the femoral level were not palpable. No blood pressure readings could be obtained in either lower extremity.

The red blood cell count was 6.03 million per cubic millimeter, and each 100 c.c. of blood contained 13.0 Gm. of hemoglobin. Urinalysis showed no abnormal findings, and

*Since this report there have been 9 additional resections for coarctation of the aorta with one death. The fatality occurred in a 35-year-old man in whom the coarctation had been excised and restoration of the lumen re-established by end-to-end union. Death was due to hemorrhage at the suture line on the tenth postoperative day. The results in the other 8 patients have been satisfactory.

the renal function as measured by phenolsulfonphthalein excretion was 59 per cent. The blood volume was within the range of normal. High voltage in the precordial leads was the only abnormality noted on the electrocardiograph, suggesting a minor degree of left ventricular hypertrophy. A roentgen film of the chest showed notching of the ribs. The retrograde carotid arteriogram demonstrated hypoplasia of the transverse arch and a definite coarctation at the junction of the aortic arch and the descending aorta. The left subclavian artery was at least twice normal size (Fig. 1).

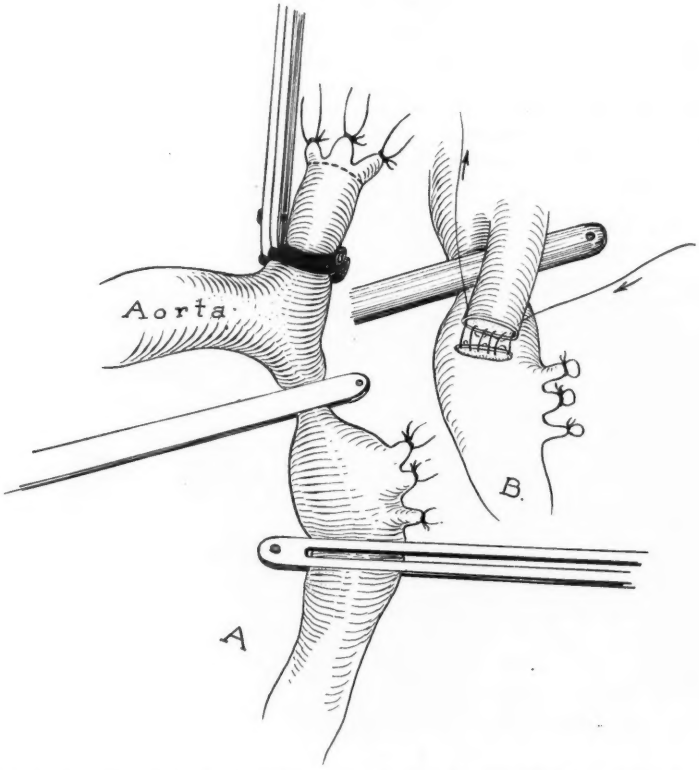

Fig. 2 (Case 1).—Several enlarged intercostal arteries were divided in order to mobilize the aorta. The subclavian artery was occluded by a Blalock clamp, the distal aorta by an aortic clamp (*A*). A small rent in the aortic wall was made at the level of the coarctation while mobilizing the aorta. This was repaired and the proximal aorta controlled by an aortic clamp until the end-to-side subclavian-aortic anastomosis (*B*) was completed.

At operation there was tremendous dilatation of the vessels of the posterolateral thoracic wall. The coarctation, located immediately distal to the origin of the greatly enlarged left subclavian artery, was not complete, as blood traversed the area to enter the distal aorta. A thrill was easily palpable at the coarcted segment. The intercostal vessels below the area of constriction were tremendously enlarged, although proximally they were of normal size. The aortic arch was hypoplastic, and slight poststenotic dilatation of the distal descending aorta was noted.

Several of the enlarged intercostal arteries were divided in order to mobilize the aorta both proximal and distal to the coarcted segment. The hypoplastic arch was bound down rather intimately so that further mobilization was deemed unwise. For this reason,

and because the coarctation was incomplete, thereby allowing some measure of blood flow, it was decided to anastomose the enlarged subclavian artery to the aorta distal to the coarctation (Blalock-Park procedure). The aorta was occluded by the application of aortic clamps proximal to and beyond the coarcted segment. The end of the subclavian artery was anastomosed to the side of the aorta with 00000 oiled arterial silk sutures laid in such a manner that the intima was everted (Fig. 2). Brisk hemorrhage which occurred from the posterolateral area of the anastomosis when the aortic clamps were removed was controlled by a single suture. The aortic clamps had been applied for fifty-five minutes.

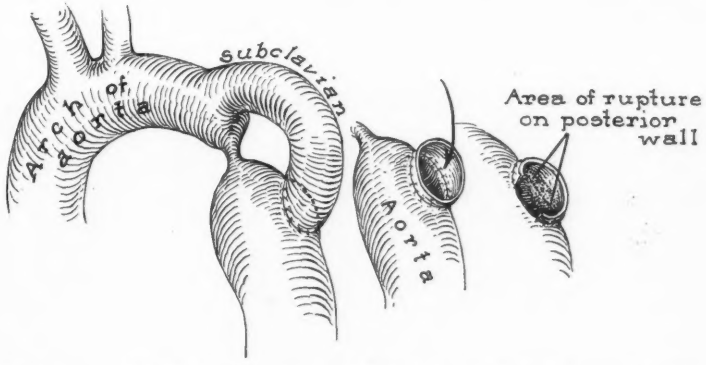

Fig. 3 (Case 1).—The anastomosis had broken down along its posterior extent. The margins of the perforation were extremely friable and contained remnants of the arterial sutures. There was but little evidence of healing.

The arterial pulsations in the lower extremities, including those of the dorsalis pedis and posterior tibial arteries, were easily palpable after operation. The peripheral arterial tension in the right arm reached 180/88 mm. Hg, while a pressure of 110 to 118 systolic was recorded in the lower extremities. During the first fourteen days following surgery, the patient was relatively symptom-free, save for the development of anemia which required the administration of 1,000 c.c. of whole blood. By that time, however, no fluid was being obtained from chest aspirations, although clinically and roentgenologically there was a large amount of fluid in the left pleural cavity. The patient began to complain of pain deep within the left hemithorax, in the left upper quadrant of the abdomen, and in the left flank. Of particular significance, on the fourteenth postoperative day, was the hemoglobin value of 75 per cent, which represented a 21 per cent decrease in a three-day period. We were unable to account for the loss of that amount of blood into the left pleural cavity. After two more transfusions and an intercostal nerve block to relieve incisional pain, his general condition improved markedly, although his temperature reached 39° C. daily. The dosage of penicillin was increased and in addition sulfonamide therapy was instituted. Repeated blood cultures produced no bacterial growth.

On the twenty-fourth postoperative day, the patient complained of progressively severe pain in the left posterior portion of the chest. There was marked tenderness in the left costovertebral angle, and although the patient appeared very pale, the hemoglobin value was 85 per cent. The urine contained no sulfonamide crystals. Since the similar episode on the fourteenth postoperative day we had been acutely aware of the probability of intrathoracic hemorrhage arising from the anastomotic line. It was our hope that conservative therapy with blood replacement would allow eventual healing of the line of anastomosis. The hematocrit values fell from 39 to 33 per cent within the next few days in spite of two blood transfusions.

A sudden shocklike state developed on the twenty-eighth postoperative day. Exploratory operation was performed immediately. The left pleural cavity was full of clotted

blood which had compressed the lung to no more than 40 per cent of its normal size. The suture line had broken down on its posterolateral aspect so that there was a defect into which one could insert the tip of the little finger. Before any attempts at repair could be made, the patient died. Cardiac massage and intracardiac adrenalin were without avail.

The margins of the perforated area were composed of extremely friable, gray tissue which was almost jelly-like in consistency. The posterior wall of the aorta at the level of the anastomosis had been displaced posteriorly by the contraction of organizing fibrous tissue in this area (Fig. 3). It is possible that the posterior retraction of the organizing fibrous tissue together with the pressure within the subclavian artery produced increasing tension on the anastomosis sufficient to cause necrosis with resulting fatal hemorrhage. The other possibility is that the line of anastomosis may have been subjected to unusual pressures and currents from the flow of blood through the intact coarcted segment.

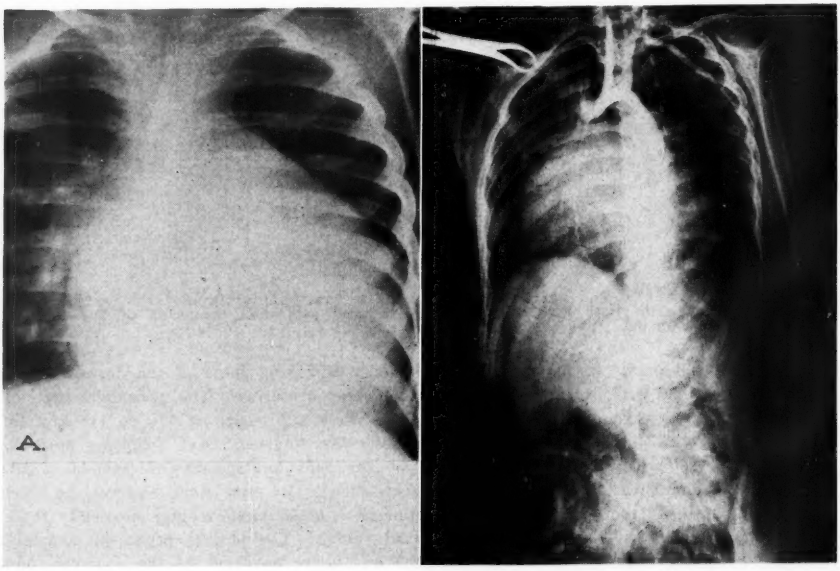

Fig. 4 (Case 2).—The plain roentgen film (*A*) showed a globular-shaped heart that was markedly enlarged with left ventricular predominance. Retrograde carotid arteriography (*B*) demonstrated coarctation of the aorta (*arrow*) at the level of the ductus arteriosus. The internal mammary artery was enlarged and the abdominal arterial trunks were well outlined.

CASE 2.—D. B., aged 4½ years, entered the University of California Hospital in May, 1947. His parents had noted easy fatigability and exertional dyspnea since the age of 15 months when the child began to walk. Five months before admission, the dyspnea and fatigue with exertion became more pronounced. Occasionally it was noted that his legs were exceptionally weak and quite cold.

On admission, the patient was undersized, pale, dyspeic, and had a rather severe though nonproductive cough. There was a moderate edema of the face, trunk, and extremities. The lower extremities were underdeveloped, the precordium was prominent, and engorgement of the neck veins was evident. The heart was markedly enlarged and a systolic murmur was heard in the left third and fourth anterior interspaces. The arterial pulsations were not palpable in the lower extremities, while those in the upper extremities exhibited normal pulsatile expansion. The blood pressure in the upper extremities was 120/78 mm. Hg; that in the legs was 106/90. Although the liver was enlarged and slightly tender, the spleen was not increased in size.

A plain roentgen film of the chest showed a globular-shaped heart that was markedly enlarged, mainly to the left (Fig. 4, *A*). The electrocardiographic tracing indicated marked right ventricular hypertrophy.

The cardiac decompensation was effectively managed by bed rest and digitalization. After sixteen days of such treatment, a retrograde carotid arteriogram was done which showed a definite constriction in the aorta just beyond the arch in the region of the ductus arteriosus (Fig. 4, *B*).

At operation the heart was enlarged, especially the left ventricle and left auricle. The area of coarctation was 1.5 cm. below the point of origin of the left subclavian artery. There was a moderate amount of poststenotic dilatation, although the aortic arch was not hypoplastic. The absence of enlarged collateral vessels was rather surprising.

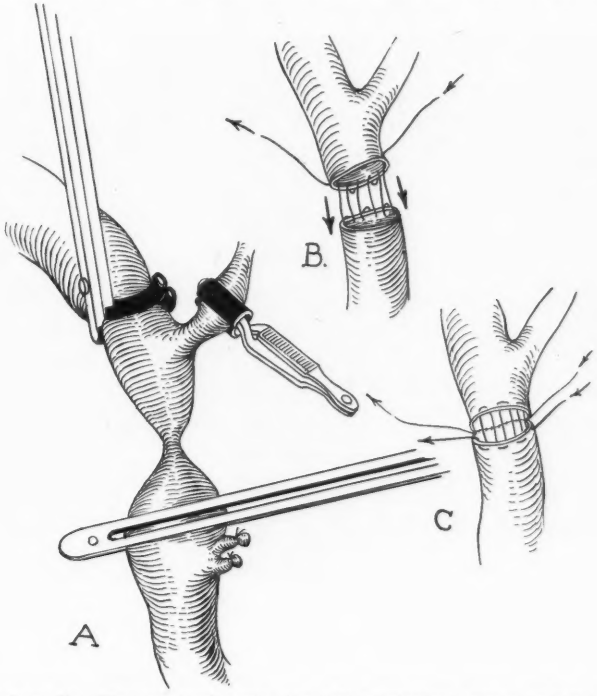

Fig. 5 (Case 2).—The proximal aorta, the left subclavian artery, and the distal aorta were controlled by appropriate clamps. After excision of the coarcted segment (*A*), an end-to-end anastomosis of the transected aortic ends was accomplished using 00000 arterial silk carried on an atraumatic needle (*B* and *C*).

The aortic arch, the left subclavian artery, and the descending aorta including the coarcted segment were mobilized. The area of coarctation was isolated by applying a Blalock clamp on the aorta proximal to the origin of the left subclavian artery and an aortic clamp similar to a Gross aortic clamp 2.5 cm. distal to the constricted area. A bulldog clamp occluded the left subclavian artery peripherally. The aorta was transected on each side of the area of coarctation and a segment 1.3 cm. in length was removed, the aortic continuity being re-established by anastomosis of the transected ends with 00000 oiled arterial silk carried on an atraumatic needle (Fig. 5). The aorta had been occluded for thirty-two minutes. The clamps were removed slowly, approximately six minutes being required for complete release. Examination of the excised segment showed a circular constriction whose aperture measured only 1.5 mm. in diameter.

The patient's postoperative course was entirely uneventful, although digitalis was administered throughout his hospital stay. The arterial pulsations in the lower extremities became normally palpable.

The boy is much more alert and active and, according to his mother, likes to do things that did not hold an attraction for him previously. The anteroposterior diameter of the chest has decreased, and he has no undue fatigue nor dyspnea. A roentgen film taken one month after surgery (Fig. 6, *A*) showed a diminution in the size of the cardiac silhoutte with changes most marked in the region of the right and left ventricles. No appreciable change was noted in the size of the left auricle. Films taken ten months after surgery showed a still greater decrease in the size of the heart, although the left ventricle and auricle were still enlarged (Fig. 6, *B*).

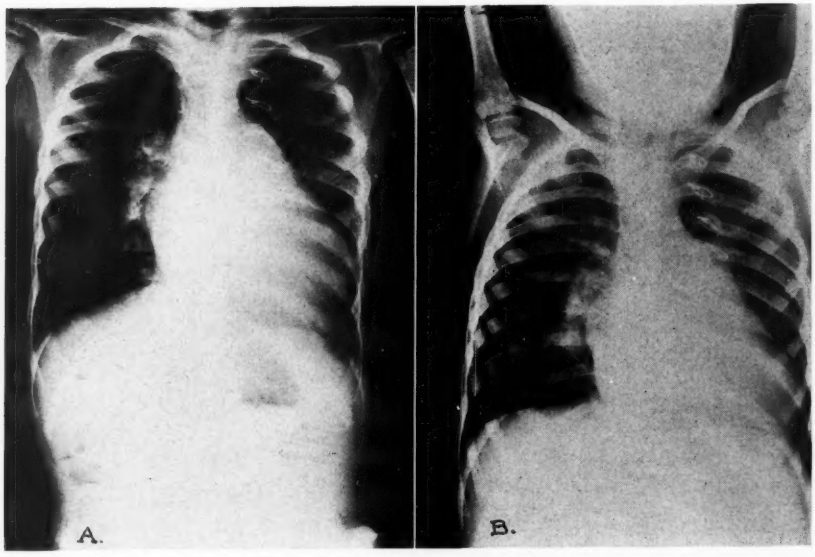

Fig. 6 (Case 2).—Plain roentgen films one month (*A*) and ten months (*B*) after operation show a progressive decrease in the size of the heart although both the left auricle and the left ventricle remain enlarged.

CASE 3.—R. M., age 13½ months, was a full-term infant who had been delivered without untoward event by cesarean section after a normal pregnancy. A heart murmur was noted at birth. He had always had rapid respirations, and for five months the parents had noted a precordial bulge. Two weeks before admission he developed progressive edema of the face and lower extremities and evidenced rather marked orthopnea.

The child weighed 16 pounds, 9 ounces, was dyspneic, and exhibited signs of cardiac decompensation with edema of the face, hands, feet, and scrotum and less marked edema of the rest of the body. The chest was symmetrical except for a slight precordial bulge. The heart was enlarged 1.5 cm. beyond the right parasternal line and reached the left anterior axillary line. The cardiac rhythm was regular. A systolic murmur was audible over the entire precordium, but was maximal in the third left interspace and was transmitted to the upper back. The liver was enlarged to the level of the umbilicus but the spleen was not palpable. The radial pulses were of good quality and the arterial tension in the upper extremities was 130/85 mm. Hg. The lower extremity pulses were less easily palpable and the arterial tension was difficult to determine reliably but was in the neighborhood of 85/70 mm. Hg.

Plain roentgen films of the chest showed marked cardiac enlargement, predominantly left sided (Fig. 7, *A*). The electrocardiographic tracing showed evidence of marked right ventricular hypertrophy.

Digitalis was administered and an adequate maintenance dose continued for a two-week period. Despite this therapy the patient continued to manifest congestive failure with orthopnea, rapid respirations, peripheral edema, and hepatomegaly. It was our opinion that the maximum effect from digitalization had been obtained and no further amelioration of the decompensated state could be expected. It was agreeable with all concerned to proceed with the diagnostic and therapeutic procedures.

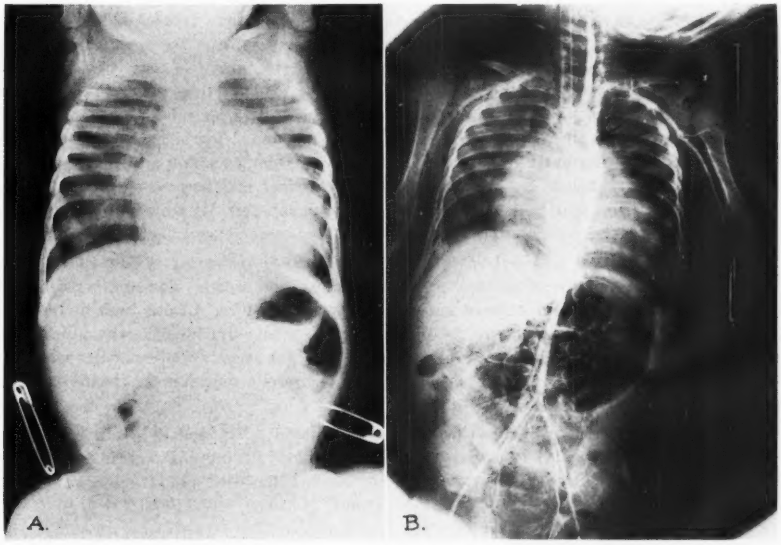

Fig. 7 (Case 3).—The plain roentgen film shows that the heart was markedly enlarged with left ventricular predominance (*A*). Retrograde carotid arteriography (*B*) verified the presence of a coarctation (*arrow*) at the level where the ductus arteriosus would be expected to enter the aorta. A moderate amount of poststenotic dilatation of the aorta was present.

Retrograde carotid arteriograms demonstrated a coarctation at about the level where the ductus arteriosus would be expected to enter the aorta. There was moderate poststenotic dilatation (Fig. 7, *B*).

Surgery was performed after twenty-seven days of preoperative preparation designed mainly to combat cardiac decompensation. The vasculature of the left posterolateral chest wall was not excessively increased or engorged. The aorta at the level of the ductus arteriosus exhibited an area of coarctation 2 cm. distal to the subclavian artery beyond which was an area of poststenotic dilatation. The aortic arch was definitely hypoplastic and a thrill was palpable in the immediate area of constriction. The distal aortic arch, the subclavian artery, and the descending aorta including the coarcted segment were mobilized by dividing the enlarged intercostal vessels and the adjacent fibroareolar tissue. Two small aortic clamps were applied proximal and distal to the coarcted segment, a 1.0 cm. segment including the constricted area was excised, and an end-to-end anastomosis between the divided ends of the aorta accomplished. The lumen of the constricted segment at the site of the stenosis measured 1.0 mm. in diameter. The aorta had been occluded for twenty-four minutes. The distal aortic clamp was removed without consequent hemorrhage, after which

the proximal aortic clamp was removed very slowly. The blood pressure fell from 110 to 60 mm. Hg (systolic) immediately after the release of the proximal clamp, but rose gradually to 95 mm. Hg.

The chest incision was closed, the dressing applied, and the endotracheal tube was about to be removed when the patient's heart ceased functioning. The usual resuscitative measures were without avail. Post-mortem examination failed to demonstrate the cause of death. In retrospect, we believe that cardiac arrest ensued because of the marked diminution of peripheral resistance produced by the removal of the constricted aortic segment. Although a full four minutes was required for the complete release of the aortic clamps, the peripheral arterial tension was not sufficiently maintained.

CASE 4.*—C. S., a 34-year-old white man, had no symptoms referable to the cardiovascular system until the age of 20 years when, while being treated for pneumonia, he was told that he possessed a heart murmur and was advised to limit his activities. However, the patient felt completely well and carried on an active athletic life, playing a great deal of tennis without difficulty. He was completely well until 1945 (aged 33 years). At that time, he first noted dyspnea while climbing stairs. Within a few months the exertional dyspnea had become sufficiently marked to require slow ascent of steps and long periods of rest after even mild exercise. By February, 1946, the patient had become markedly dyspneic even with mild exertion and noted an occasional aching substernal pain. Two months later nocturnal dyspnea appeared and walking on level ground became extremely difficult.

The common carotid and subclavian arteries pulsated vigorously. The radial pulses were prominent but a marked lag in the femoral pulses was noted. The ankle pulses were faintly palpable on the right but were not discernible on the left. Bruits were audible over the left anterior and posterior thoracic wall, the latter as far inferiorly as the ninth dorsal segment. The heart was moderately enlarged and there was a systolic thrill which was maximal in the left third interspace. The harsh aortic systolic murmur was heard best over this same area and was transmitted posteriorly.

The plain roentgen film of the chest showed a dilated aorta and notching of the ribs. The electrocardiogram revealed left ventricular hypertrophy. Retrograde carotid arteriograms were suggestive of coarctation but were not conclusive. Repeated blood cultures failed to reveal bacterial growth.

At operation, the arterial supply of the posterolateral chest wall was greatly increased and the individual vessels were enlarged and tortuous. The collateral arterial circulation within the left hemithorax was enormous, especially the fourth and fifth intercostal arteries which measured about 1.0 cm. in diameter at their junction with the aorta. The internal mammary artery was huge and had a forceful pulsation. The upper three pairs of intercostal arteries were divided and ligated. The aorta at the level of the coarctation was narrowed to a diameter of approximately 1.5 cm. It was moderately dilated above and below. The aorta was occluded proximal to the subclavian artery and distal to the coarcted segment while the subclavian artery was occluded distally. The coarcted segment was resected. The aperture at the site of the stenosis was only 2 mm. in diameter. The intima of the aorta above the level of the coarctation exhibited definite yellow patches of arteriosclerosis and that below the level of the coarctation was somewhat friable. An end-to-end anastomosis of the transected aortic ends was accomplished with no little difficulty. The aorta had been occluded for eighty minutes. The aortic clamps were removed slowly, three minutes elapsing for the removal of the proximal aortic clamp. During release of this clamp, an infusion of whole blood was administered into the right radial artery. The infusion was given rapidly because the arterial pressure fell rapidly to as low as 40 mm. Hg, at which time the cardiac rhythm was extremely irregular, even exhibiting momentary arrest. By cardiac massage and reapplication of the aortic clamps, regular cardiac rhythm was re-established. However, with the maintenance of adequate cardiac activity, considerable hemorrhage occurred from the

*This case is being reported through the kindness of Dr. Norman Freeman, who studied and operated upon the patient.

anastomotic line. This was controlled by the application of a pledget of oxidized cellulose directly to the suture line.

The postoperative course was relatively uneventful until the eleventh postoperative day when the patient suddenly complained of severe left precordial pain, rapidly became comatose, and died within a few minutes. Necropsy examination showed but little healing of the suture line. About 0.5 cm. proximal to the line of anastomosis was a small perforation in the aortic wall through which extensive hemorrhage had occurred. The transverse aortic arch was atherosclerotic and the aortic valves were stenotic. The cerebral vessels were also atherosclerotic.

CASE 5.—E. B., a 19-year-old white man, was known to have had hypertension since the age of 15 years, when he consulted a physician because of headaches and occasional dizziness. There was no progression of symptoms until six months before admission, when he noted increased fatigability, more frequent headaches, several episodes of faintness with blurred vision, exertional dyspnea, and palpitation. He had not been aware of ankle edema, orthopnea, or chest pain.

On examination, prominent intercostal arterial pulsations could be palpated over the posterior mid-chest. The heart was not enlarged but a loud systolic murmur could be elicited both over the precordium and over the left posterior part of the chest. The arterial tension in the upper extremities measured 180/102, while a value of approximately 94/60 was obtained in the right leg. The blood pressure in the left leg was not obtainable. The femoral pulsations were bilaterally present but extremely weak and the arterial pulses at the foot and ankle were not palpable.

A plain roentgen film of the chest showed that the heart was moderately enlarged and that the increase in size was predominantly left ventricular. Minimal notching of the ribs posteriorly was noted, and the aortic knob was absent. The electrocardiogram was indicative of left ventricular hypertrophy. The venous pressure measured 13.5 cm. of water. Intravenous urograms revealed no abnormal findings.

Imperfect visualization of the aorta was obtained by the retrograde carotid arteriograms, but there was strong evidence from these studies that a coarctation was present immediately distal to the origin of the left subclavian artery.

At operation the coarctation was located 1.0 cm. distal to the point where the left subclavian artery emerged from the aorta. Many enlarged collateral vessels entered the aorta distal to the coarcted segment, although there was no poststenotic dilatation. The left subclavian artery was considerably enlarged. This vessel, along with the transverse arch and that portion of the aorta containing the coarcted segment, was completely mobilized. The proximal aorta was occluded by a Bethune lobectomy clamp and the aorta distal to the constricted segment by a straight aortic clamp. The aorta in the area of coarctation was transected and the proximal end closed by a running 00000 Deknatel silk suture. An end-to-end anastomosis between the subclavian artery and the distal aorta was then accomplished (Fig. 8). The occluding aortic clamps were in place for seventy minutes. Prior to the release of the aortic clamps, pneumatic cuffs previously applied to both lower extremities were inflated to prevent pooling of the blood in the legs (Fig. 9). After the aortic clamps had been slowly removed, the pneumatic cuffs were released at fifteen minute intervals. There was no change thereafter in either the cardiac rhythm or the blood pressure recordings.

The femoral and pedal pulses were easily palpable immediately after surgery and have maintained their quality. The arterial tension in the upper extremities has remained elevated in the neighborhood of 180/100 mm. Hg. and that of the lower extremities can now be recorded at 190/120. It is still too soon after surgery (three weeks) to attempt to foretell whether or not the existent hypertension will be maintained.

CASE 6.—R. H., aged 6½ years, had had frequent upper respiratory tract infections and had gained weight poorly since birth. For two years before entry his parents had noted that he had increased fatigability and for the previous six months he had almost daily severe headaches. Four months before admission a heart murmur was discovered but its etiology could not be determined.

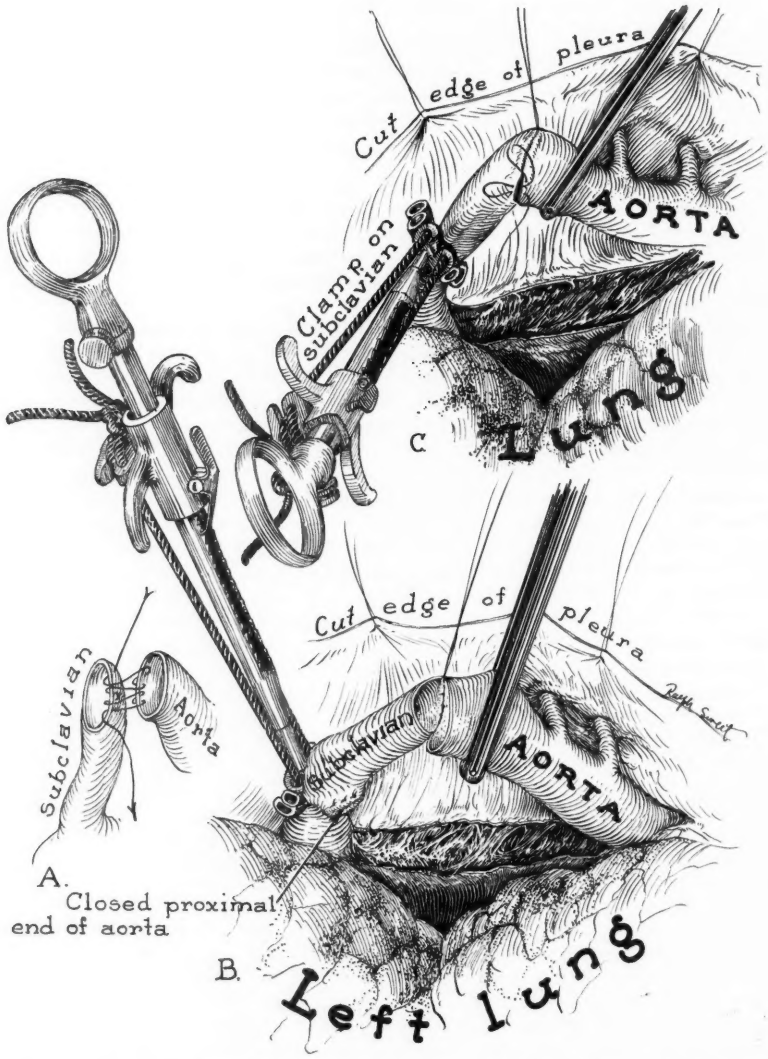

Fig. 8 (Case 5).—The aorta was occluded proximally by a Bethune lobectomy clamp, distally by an aortic clamp. The aorta in the area of the coarctation was transected and the proximal end closed (*B*). An end-to-end subclavian-aortic anastomosis was performed. The posterior suture line was first applied (*A*), after which the anterior row of sutures was laid to complete the anastomosis (*C*).

The child was somewhat small for his age and in comparison to the parental stock. A transmitted systolic murmur could be heard along the carotid arteries, more so on the left than on the right. A systolic murmur was present over the entire precordium but was most audible in the second interspace just to the right of the sternum. The murmur was transmitted both to the back and into the left axilla. The arterial tension in the left arm was 140/75 mm. Hg, while neither a pressure nor a pulse was obtainable in the right arm. The femoral and pedal pulses were likewise not palpable, nor could a blood pressure reading be determined in the lower extremities.

The plain roentgen film of the chest showed a small notched defect on the inferior border of the left fifth rib. The heart was not enlarged. During the first attempt to perform contrast angiography, considerable Diodrast solution extravasated into the periarterial tissues as far inferiorly as the transverse arch, obscuring the detail in this region to such an extent

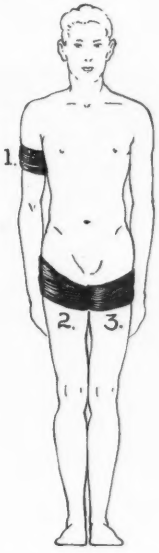

Fig. 9.—Pneumatic cuffs about the proximal portions of the extremities serve both to prevent pooling of blood in the large vascular beds and to maintain an increased resistance when the aortic clamps are removed.

that it was necessary to repeat the procedure. A second series of arteriograms demonstrated beyond doubt the presence of coarctation of the aorta at the beginning of the descending portion of the aortic arch (Fig. 10).

At operation there was considerable reaction about the aortic arch due to the previously extravasated Diodrast solution. This made the operative procedure more difficult than would have otherwise been the case. The coarctation was located at the level of the obliterated ductus arteriosus. The left subclavian artery was very large and emerged from the parent vessel immediately proximal to the area of coarctation. A large anomalous vessel approximately one-half the size of the left subclavian artery joined the aorta immediately proximal to the coarctation.

The transverse aortic arch, the left subclavian artery, and the aorta distal to the coarcted segment were mobilized as completely as justifiable in the presence of the previously mentioned inflammatory reaction. Because of the proximity of the coarcted segment to the subclavian artery, an end-to-end anastomosis between the divided ends of the aorta was quite impossible. The proximal aorta was occluded by a Bethune lobectomy clamp, the distal aorta

by a straight aortic clamp, and the anomalous vessel by a bull-dog clamp. An end-to-side anastomosis was then accomplished between the end of the subclavian artery and the side of the aorta distal to the coarcted segment. The occluding aortic clamps had been in place for forty-five minutes. The area of constriction was not excised (Fig. 11). Before removal of the aortic clamps, pneumatic cuffs previously applied to the lower extremities were inflated to prevent pooling of blood in the legs. The aortic clamps were removed slowly, after which the pneumatic cuffs were released individually at fifteen minute intervals. No cardiac irregularity or significant change in blood pressure readings occurred.

At the conclusion of the operative procedure, the pedal pulses were found to be strong and bounding and have remained so. The tension in the right arm now measures 120/100, while the readings in the lower extremities are 152/110 in the right leg and 160/110 in the left leg, seven weeks after surgery.

On the patient's tenth postoperative day, a marked weakness of the musculature of the left lower leg was noted and shortly thereafter some weakness of the muscles of the right

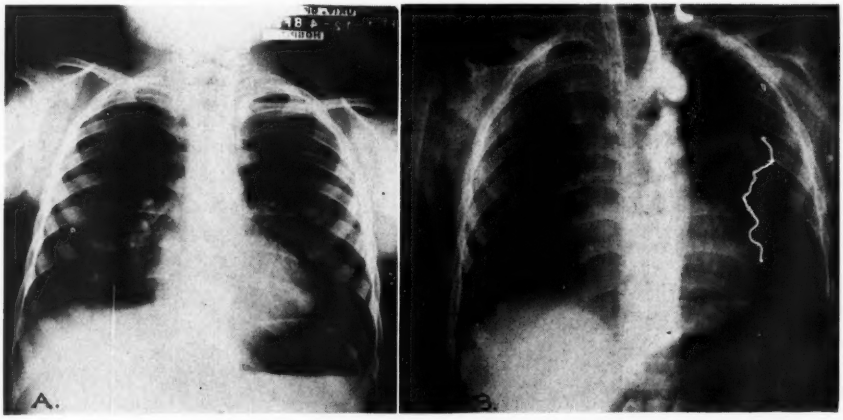

Fig. 10 (Case 6).—The plain roentgen film showed a small notched defect on the inferior border of the left fifth rib. The heart was not enlarged (*A*). Retrograde carotid arteriography showed a definite coarctation (*arrow*) in the first portion of the descending aorta (*B*).

posterior cervical region was thought to be present. Because of these complications the postoperative hospital stay was prolonged (seven weeks). The course was otherwise entirely uneventful and, moreover, the opportunity was presented for observing the metamorphosis of an inactive, introspective, shy, and easily fatigued child into one who shares rather marked activity with the other little patients on the ward.

DISCUSSION

It has been mainly through the efforts of Crafoord in Sweden and Gross in America that the surgical attack upon coarctation of the aorta has achieved success. This particular realm of surgical endeavor, though still in its infancy, has offered new hope and promise to those unfortunate people afflicted with the anomaly. Although there are no doubt many cases still unreported in the literature, an analysis of those reported through March, 1948, is of interest. Table I indicates that most of the operations have been performed by Gross and Crafoord. Of the forty-five patients operated upon in the hope of carrying the procedure to its completion, forty were subjected to some form of excision

and/or anastomosis. In this group of forty patients, there were nine deaths, a mortality of 22.5 per cent. It is reasonable to assume that when a larger number of cases has been reported, the mortality rate will approximate that of other surgical procedures of similar magnitude.

The collected data are in no sense complete, but it is apparent that the majority of deaths occurred in those patients who had reached adulthood. This does not signify that age is an absolute contraindication to operation, but rather emphasizes the fact that a guarded prognosis must be made when

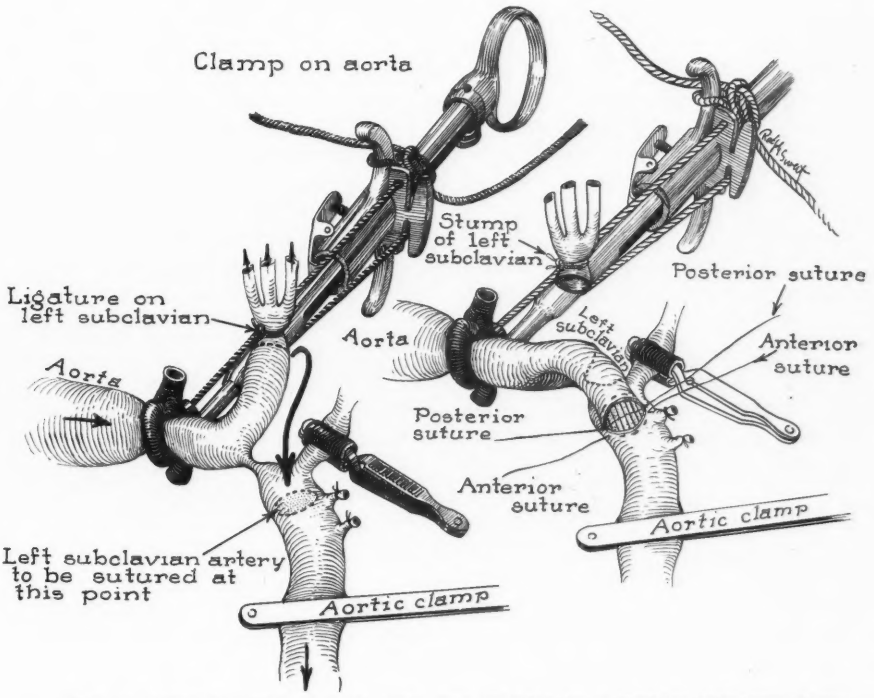

Fig. 11 (Case 6).—The proximal aorta was occluded by a Bethune lobectomy clamp, the distal aorta by an aortic clamp. A large anomalous vessel sprung from the posterolateral portion of the aorta immediately distal to the coarctation and was controlled by a bulldog clamp. An end-to-side subclavian-aortic anastomosis was accomplished without resection of the constricted segment.

operation is the only hope of salvation for these people. Considering the magnitude of the operative procedure and the danger of working with vessels of aortic caliber, the fact that good results are obtained in 62 per cent of cases is gratifying.

That four of the nine deaths occured from postoperative hemorrhage is not surprising. Two others occurred from marked interference with cardiac function, probably occasioned by the release of peripheral arterial resistance due to removal of the constricted segment. The remaining causes of death appear to be related both to the consequences of operation upon large blood

TABLE I.—RÉSUMÉ OF REPORTED OPERATIVE CASES IN THE WORLD LITERATURE

AUTHOR	EXPLORATION (NUMBER OF CASES)	OPERATION (NUMBER OF CASES)	DEATHS	CAUSE OF DEATH	AGE OF PATIENTS WHO DID NOT SURVIVE	ANESTHESIA	NUMBER OF GOOD RESULTS
Gross	18	15	2	1. Cardiac dilatation and irregularity upon release of aortic clamps	5 yr.	Cyclopropane	12 patients of 15 (2 deaths, 1 hypertension not relieved because of unsatisfactory anastomosis)
				2. Death 24 hr. postoperatively, probably from hemorrhage from suture line	30 yr.		
Crafoord	17	15	2	1. False aneurysm at aortic suture line	Unknown	Cyclopropane and nitrous oxide	11 of 15 (2 deaths, 2 inoperable)
				2. Postoperative hemorrhage from large intercostal vessel (anastomosis intact)	Unknown		
Clagett	1	1	0			Not noted	1 (age of patient, 34 yr.)
Jaehning	3	3	2	1. Massive atelectasis of left lung, and pulmonary edema of right lung	16 yr.	Not noted	1 of 3 (2 deaths)
				2. Atelectasis of left lung, and obstructive emphysema of right lung	21 yr.		
Stephens and Grimes	6	6	3	1. Postoperative hemorrhage at suture line	16 yr.	Nitrous oxide and curare	3 of 6 (3 deaths)
				2. Postoperative hemorrhage above suture line	34 yr.		
				3. Probable cardiac irregularity and dilatation	13½ mo.		
Total	45	40	9 (22.5%)	1. Hemorrhage postoperative, 4 2. Cardiac irregularity, 2 3. Atelectasis and edema, 2 4. Aneurysm at suture line, 1			28 (62.2%)

vessels and to the magnitude of the procedure and its profound alteration in cardiovascular-respiratory physiology.

The six cases reported here constitute but a fraction of those reported to this date in the literature. However, it seems to us that all available experience with the surgical treatment of coarctation of the aorta should be pooled for the mutual benefit of all. The results of the experiences at the University of California Hospital are briefly summarized in Table II. End-to-end anastomosis was performed in three cases, an end-to-side anastomosis between the left subclavian artery and the aorta without excision of the constricted segment in two cases, and an end-to-end anastomosis between the end of the subclavian artery and the distal aorta after excision of the area of coarctation was done in one case. There is general agreement that an end-to-end union is the treatment of choice whenever possible. The site of origin of the coarctation is the main factor which determines the type of procedure to be done. If the coarcted area is situated close to the point where the subclavian artery emerges from the aorta, resection with end-to-end anastomosis is contraindicated because of the distance between the two aortic segments. Our experience with Case 1, in which the suture line of the end-to-side subclavian-aortic anastomosis broke down, suggests that an end-to-end union between the distal transected aorta and the subclavian artery might offer distinct advantages over the so-called Blalock-Park procedure which leaves the coarcted area intact.

Many authors feel that few, if, any, of the intercostal arteries should be sacrificed since they form collateral vascular channels. It is probable, however, that the danger of complicating or fatal hemorrhage from an inadequately controlled aorta is far more serious than is the sacrifice of a minor area of collateral vessels. In addition, the constrictionless aorta assumes all the blood transport previously carried by the collateral vasculature.

In two cases, hemorrhage from or near the suture line led to fatal termination. In one of these, even though the suture line showed but little evidence of healing at necropsy, the site of exsanguinating hemorrhage was well above the anastomosis. It is reasonable to assume that the aortic perforation occurred from the trauma of the occluding aortic clamp in an area exhibiting atherosclerosis. In the other case, perforation occurred at the suture line, presumably from excessive tension or from abnormal influences upon the suture line resulting mainly from the presence of the open coarctation.

The one fatality on the operating table exhibited no direct cause of death at autopsy. It seems likely that the child died from the effects of acutely disturbed cardiovascular physiology. One would expect immediate evidence of cardiac failure when there is a sudden decrease in peripheral resistance, yet in this child cardiac failure did not occur until approximately thirty minutes after the aortic clamps were released. This operative mortality might have been prevented by a more gradual decrease in peripheral resistance. The application of pneumatic cuffs on the lower extremities, maintaining the cuffs at pre-resection pressure levels, and the release of the individual cuffs at fifteen minute intervals after the removal of the aortic clamps, might have provided an adequate mechanism to accustom the heart to a new level of

TABLE II. RÉSUMÉ OF CLINICAL AND OPERATIVE DATA, UNIVERSITY OF CALIFORNIA HOSPITAL

AGE	SEX	LOCATION OF COARCTATION		OPERATIVE PROCEDURE			SEQUELAE	TIME AORTA OCCLUDED (MINUTES)
		1.5 TO 2.0 CM. DISTAL TO ORIGIN OF SUBCLAVIAN ARTERY	IMMEDIATELY DISTAL TO ORIGIN OF SUBCLAVIAN ARTERY	RESECTION WITH END-TO-END ANASTOMOSIS	END-TO-SIDE ANASTOMOSIS BETWEEN SUBCLAVIAN AND AORTA	END-TO-END ANASTOMOSIS OF SUBCLAVIAN ARTERY TO DISTAL AORTA WITH EXCISION OF COARCTATION AND CLOSURE OF ITS PROXIMAL END		
13½ mo.	M	X		X			Operative mortality	24
4½ yr.	M	X		X			Well	32
6½ yr.	M	X			X		Well	45
16 yr.	M		X		X		Hospital mortality	55
19 yr.	M		X			X	Well	70
34 yr.	M	X		X			Hospital mortality	80

peripheral resistance. Particularly is this true in the younger age groups. Rapid whole blood infusions while the aortic clamps are being removed accomplishes much the same thing.

At this moment there is nothing to offer those unfortunate people with areas of narrowing, usually in the descending aorta, which are of such length that it is impossible to re-establish continuity by an appropriate anastomosis. Save for this unfortunate contingency, there is no absolute contraindication to the surgery of coarctation of the aorta. Advanced age with its attendant arteriosclerotic changes, especially in the proximal aorta, in combination with other factors, may constitute an absolute contraindication in the specific case, but perhaps only relative reasons against operation in others.

The condition of the proximal aorta is all important. A dilated, attenuated, and weakened proximal aorta whose intima is studded with arteriosclerotic patches is likely to hold sutures poorly. It is generally agreed that in many cases, although poor results may be expected, surgery must be performed as the only hope for salvation of the patient. It is emphasized that one can expect better results in the child or young adult in whom degenerative changes have yet to appear.

SUMMARY

In the adult type of coarctation, a sharp constriction or obliteration of the descending aorta occurs at or near the insertion of the ductus arteriosus. The aorta proximal to the coarctation is especially likely to develop inflammatory or degenerative changes, the exact cause of which is unknown. There is no doubt that coarctation of the aorta shortens the life of the individual. Cardiac decompensation, spontaneous rupture of the aorta, cerebral accidents, and mycotic endarteritis are the most frequent causes of death.

Six patients exhibiting coarctation of the aorta have been operated upon at the University of California Hospital. Three patients survived. One fatality was operative; two others, from exsanguinating hemorrhage, occurred in the immediate postoperative period. One of these resulted from necrosis at the line of anastomosis; the other, although showing but little evidence of healing at the suture line, had a small perforation proximal to the line of anastomosis. The opening apparently had occurred in an area of atherosclerosis traumatized by an occluding aortic clamp.

The operative death, in retrospect, was probably a preventable one. If the peripheral arterial resistance and blood volume had been maintained until the cardiovascular system had had sufficient time to adjust to the new pressure relationships, normal cardiac function would probably have ensued.

A brief review of the literature indicates that an over-all mortality rate of 22.5 per cent exists for the surgery of coarctation of the aorta. Most of the reported deaths have occurred in patients in older age groups and the most common cause of death has been hemorrhage, usually at the line of anastomosis. It is quite generally agreed that an end-to-end aortic anastomosis is the procedure of choice.

There are probably no absolute contraindications to the surgery of the adult type of coarctation of the aorta. In most instances, surgery remains the only hope for those even in whom the operative risks are great. Within the realm of reason, these patients should be offered the benefit of operative therapy.

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DISCUSSION

DR. ROBERT E. GROSS, Brookline, Mass.—There are many things I would like to discuss in Dr. Stephens' paper but I shall limit my remarks to treatment of cases which have been proving most difficult for me. In a series of forty-five patients, I decided not to operate upon four because of conditions which I thought were inoperable at that time. Of the forty-one patients in whom resection was done and end-to-end anastomosis performed, there have been six deaths and I think three of those would be preventable with present day technics. In this series with resections there have been three disappointments; in one there has been no lowering of pressure in the arms, and in the other two there has been only moderate lowering; without question the anastomoses in these patients were all unsatisfactory, and the opening obtained in each case was too small.

Considering these three, plus the four not operated upon, we are searching for some new surgical technique for treatment of patients who have very long areas of narrowing, or who have very rigid vessels, and in whom it is impossible to get the ends together easily after a piece of aorta has been removed; we would like to have some sort of graft to put in a gap of this sort. Working with dogs it has been possible to show in a series of almost seventy animals, with varying types of preservation of vessels, that one can remove arteries from dogs, preserve them for periods of time, and implant them into recipient animals and

have them live. The method that we have finally decided upon is one in which the artery is kept in a complicated electrolyte solution with seven or eight substances in it; it contains glucose for nourishment; it also contains serum homologous to that animal. It is possible to show that vessels kept in this way in the cold at 1 to 4° C. have tissues which are living. We have made tissue cultures, more than one thousand of them, taking pieces of tissue every few days from these vessels which have been kept in the cold, to determine how long the specimens of aorta are viable. In short, it is possible to show that an artery can be kept alive for thirty to forty days. We have kept vessels by this method for such periods and then implanted them into recipient dogs and have kept such dogs for as long as a year, and have made very good aortic channels by such grafting. In no instance have we had any dehiscence of a suture line and in no instance have we had death of an animal from thrombosis of one of these grafts. We have had a few minor thromboses, with very little diminution of the size of the lumen of the vessel.

(Slides) We have been preserving sections of arteries from people who have died in automobile accidents, keeping them for a period of one month or more. I have implanted eight such segments into patients in whom we were attempting to make some sort of anastomosis on the Blalock principle between the aortic and pulmonary systems, placing them in different areas to bridge such a gap. Two of the patients have died; one from cardiac decompensation in two days; the other died twelve days later from the effects of Dicumarol therapy, with bleeding from various parts of the body. Those patients both had viable and patent grafts at autopsy. The remaining six patients have had the grafts in place for as long as four months, they have continuous murmurs which they did not have before, and they have had amelioration of the cyanotic state.

Carrying this graft technique to patients with coarctation of the aorta, which is the subject of today's interest, I have used a graft in only one subject. (Slide.) This sketch was made at the time of operation; a graft 5 cm. in length was used; this graft had been preserved for thirty days. This patient has had complete alleviation of hypertension; the arm pressures are normal and the leg pressures are 30 to 35 mm. Hg above that in the arms; in short, there is evidence that we have a pathway which is adequate. Whether this will be the answer to the problem of bridging gaps in those patients who have a rigid aorta or who have a long area of constriction I do not know. We mention this merely as an indication of the lines along which we are working.

DR. J. C. JONES, Los Angeles.—We have operated upon thirteen patients for coarctation of the aorta and have completed some type of anastomosis in all. The end of the subclavian was anastomosed to the side of the distal aorta in two patients. The coarctation was resected and the end of the subclavian was anastomosed to the end of the distal aorta in two instances, and in nine patients the coarctation was resected and an end-to-end anastomosis of the aorta was completed. The ages of these patients ranged from 2½ to 31 years.

There were two deaths in the series. One was a 31-year-old man with a large heart, who had been decompensated for two years. He had advanced arteriosclerosis and, after we were able to complete a satisfactory anastomosis, the posterior wall of the aorta at the site of the proximal clamp developed a leak on removal of the clamp. This was repaired with some difficulty but he died suddenly on the ninth postoperative day of a dissecting aneurysm, well proximal to the anastomosis which was intact at autopsy. The other death was a male of 17 years in whom we had no difficulties in resecting the coaretation and completing an end-to-end anastomosis, but he died suddenly four hours after operation, presumably of a leak or rupture at the site of anastomosis. The seven survivors of the resection with end-to-end anastomosis of the aorta have had excellent results, with blood pressures in their legs as high as, if not higher than, in the arms. The two patients in whom the end of the subclavian was anastomosed to the end of the distal aorta likewise have had excellent results. The two patients in whom the end of the subclavian was anastomosed to the side of the distal aorta are improved, but the results might be estimated as only fair.

We have operated upon three patients of 5 years or younger; one was 2½ years old and his, incidentally, was the easiest operation from a technical point of view; he had the smoothest postoperative course and he was able to go home on the tenth day after operation. Since the collateral circulation is well developed early in life in these individuals the indications are to operate upon them early. We have employed a variety of methods in our end-to-end anastomoses of the aorta, but at present prefer the use of four everting stay sutures combined with four running over-and-over sutures in the four quadrants.

DR. ROLLINS HANLON, Baltimore.—On Dr. Blalock's service at The Johns Hopkins Hospital twenty-four patients with coarctation of the aorta have undergone exploratory thoracotomy. The age of the patients ranged from 6 to 34 years, with the majority over 20 years.

There have been three deaths in the series. One resulted from a technical error, one from a cerebral vascular accident, and one from multiple associated cardiac abnormalities. The latter two deaths were in children. In one instance resection of the coarctation was not possible. Twenty patients underwent successful resection of the coarctation. In three continuity of the aorta was re-established by anastomosis of the subclavian artery to the distal aorta. In seventeen an end-to-end anastomosis was carried out between proximal and distal aorta. This is the procedure of choice.

The anatomic findings in one instance are of sufficient interest to warrant presentation. (Slide.) This 20-year-old girl had a coarctation proximal to the origin of the left subclavian artery—an abnormality which had been diagnosed by clinical studies including angiocardiography. There was notching of the ribs on the right but not on the left. There was a large left carotid artery and a somewhat smaller left subclavian artery with the intervening vertebral artery arising directly from the aorta instead of from the subclavian artery. The left radial pulse was unaffected by occlusion of the left subclavian artery, indicating excellent collateral circulation. The blood pressure in the left arm was lower than that on the right before operation, and this relation persisted after satisfactory aortic anastomosis, with the left subclavian artery divided. The subclavian artery was sacrificed since it would have interfered with an end-to-end aortic anastomosis. In resecting the coarctation, in order to have the proximal opening comparable in size to that in the distal aorta, it was necessary to extend the incision up the lateral wall of the vertebral artery. (Slide.) The completed result was quite satisfactory, and the patient remains well more than three months after operation.

In closing, three technical points may be mentioned. First, an everting mattress suture is used. Second, the collateral circulation, particularly the intercostal arteries, has been preserved if at all possible. Finally, a special clamp similiar to the Potts' clamp is used to occlude the proximal aortic segment. (Slide.) This allows blood to flow freely up the subclavian artery while occluding the aorta just distal to it, thus providing adequate working length with increased safety even in the very short proximal segments that are encountered not infrequently.

DR. CLARENCE CRAFOORD, Stockholm.—From the excellent result we have just heard from Dr. Blalock and his co-workers, and from my own experience, I think we may state that radical treatment of coarctation of the aorta can no longer be considered as a surgical equilibristic procedure; I think it can be said that it is a sound surgical procedure. The results I have had support the conclusions which can be drawn from Dr. Blalock's presentation.

We have now operated on thirty-two patients with coarctation. resection was done in thirty-one of those and in one case we did a "Blalock operation" with anastomosis, end-to-side, between the subclavian artery and the aorta below the coarctation. In this series of thirty-two cases we had two postoperative deaths, one due to acute bleeding on the afternoon of the day of operation, the other due to formation of a dissecting aneurysm which broke into the bronchial tree, from which one month after operation the patient died from massive hemoptysis. In addition to the thirty-two cases operated upon, exploration has

been done in five cases, intending to try radical operation, but conditions were such that an anastomosing operation was not possible. These patients were in the older age group, between 25 and 35 years of age. In all, arteriosclerotic changes of the walls of the arteries were present, which made any kind of surgical intervention on the vessels impossible. In that group of five cases in which exploration and suture was done again we had two deaths shortly after operation. Because of that I would like to point out the value of a method of investigation that we have developed recently to try to make as accurate an anatomic diagnosis of the lesion as possible before operation. We have introduced an ordinary cardiac catheter through the radial artery, pushing it forward, down into the aortic arch. With rapid injection of 50 c.c. of 70 per cent Diodrast we can get an excellent x-ray picture of the first part of the aorta and the coarctation. I hope that as we become more familiar with this method of investigation we may be able to exclude most of the unsuitable cases from exploratory thoracotomy and select in advance the ones on whom we can hope to achieve a good result.

(Slides.) In this picture can be seen the catheter passed down into the arch of the aorta to the level of the aortic ostium. The curved outlines of the aortic valves are shown, and also a fairly good picture of the coronary arteries. We hope to be able to carry out studies on the coronary arteries by this means in the future. In the cases which this procedure had been carried out we have had no reaction which contraindicates a continuance of the method.

(Slide.) This case was rather difficult to interpret; it was one of the first upon which this investigation was done and we did not know how to interpret these different shadows. We therefore explored to confirm our x-ray findings, and in this drawing you can see what was found. There were multiple aneurysms on the aortic arch which made radical procedure of any sort impossible.

We have been able to follow twenty-seven of the thirty-two cases for a period of time ranging between three months and three and one-half years. Of the remaining five patients, two have died in the immediate postoperative period as just mentioned and three have been so recently operated upon that it is too early to tell how they will progress. In thirteen of the twenty-seven cases the blood pressure has returned to normal, with higher pressures in the legs than in the arms. In six, the blood pressure is equal in the legs and the arms and in eight, it is still slightly higher in the arms than in the legs. In these eight we have, from an anatomic viewpoint, not been able to perform a sufficiently wide anastomosis.

In three cases of the thirty patients surviving we have had development of small postoperative dissecting aneurysms, demonstrated by direct aortography after operation. Two of the three are symptomless; they have not been growing. In one case there is sign of increase in the size of the aneurysm, the patient has symptoms, and we are discussing the possibility of reoperation.

The principal difference between the technique described by Blalock and Gross and the one I use, is that both Blalock and Gross use everting sutures. I have felt that one should try to resect as much as possible of the constricted area which is nearly always so long that it is difficult to resect enough. Because of that I have used an anatomic suture with coaptation of the different layers of the aortic wall against each other, which permits resection of a few millimeters more than if an everting suture is used.

I have also preferred this suture because I believe that the healing will be better and stronger when the different anatomic layers of the aorta are in direct contact. I do not know whether Dr. Blalock and Dr. Gross have had postoperative aortograms made, or if they have found any postoperative symptomless aneurysms.

A comparison between our series in that respect might give some indication as to whether the anatomic or the everting suture is to be preferred.

The mean blood pressure before operation in my cases has been 180/100 in the arms and about 100 mm. of mercury systolic in the legs. After operation the pressure in the arms has gone back to normal as mentioned previously.

DR. OSLER ABBOTT, Atlanta.—In our more recent coarctation operations we instituted arterial transfusion in the arm on the uninvolved side to be ready at a set level of blood pressure at the time of release of the anastomosis. The fall of pressure in these patients has been to the level set on the manometer and has not gone below that level. I believe that arterial transfusion has a tremendous place at this stage of the operation. It is also interesting that this method of transfusion was recently used on the urological service at the time of removal of an adrenal chromocytoma with no drop in blood pressure.

The other factor I would like to mention is the occurrence in our series two years ago of encountering a patient in whom a large patent ductus was present, and as soon as the ductus was occluded with a rubber shod clamp, intractable auricular fibrillation occurred, which could not be corrected by any method we attempted. On releasing the patient from oxygen it became obvious that there was a third deformity, namely, a hidden pulmonary stenosis; we did not resect the coarctation.

I would also like to mention the one death in our series occurring when we tried to form an artificial arch in a patient having only two major vessels coming off the arch, namely, a combined innominate and left common carotid and a left subclavian; the coarctation was proximal to the left subclavian, and the subclavian wall was used for an artificial construction of the arch. The patient died eighteen hours after operation when the pressure rose to 134. I am certain that if we had reinforced this graft we might have been successful.

DR. CLARENCE CRAFOORD, Stockholm.—There has been some talk about the rapid fall of blood pressure caused by rapid releasing of the aortic clamps. This is difficult for me to understand. I have had the aorta crossclamped in altogether seventy cases of coarctation and patent ductus, and have never taken any precaution in releasing them slowly, with no blood-pressure falls of any significance in connection with removal of the clamps. In the ductus cases where we have no collateral circulation, when aortic clamps are applied the blood pressure rises sometimes to 200 mm. of mercury or more. The blood pressure has dropped to the previous level after the clamps have been released. I do not think the explanation given by Dr. Gross is the correct one, that it is the releasing of the clamps that is the cause of the blood pressure fall; I am convinced there is some other factor such as unsatisfactory ventilation during the anesthesia, for all these patients as far as I know are allowed to breathe themselves during the operation and have not been under controlled respiration. I am convinced that if adequate ventilation has been used the small additional stress to which the heart is subject on removal of the aortic clamps will not affect the heart at all.

There is a point I would like to stress when giving large amounts of blood rapidly. I believe it is better to give fresh blood from heparinized donors, than to give old blood. Fresh blood will function much longer than stored blood from a blood bank.

THE SURGERY OF PULMONARY STENOSIS

Experiences With Left Subclavian to Left Pulmonary Artery Anastomosis

Emile Holman, M.D.
San Francisco, Calif.

IN A DISCUSSION of Cutler's[1] startling, though unsuccessful attempts to enlarge the stenosed mitral valve by operation, Harvey Cushing in 1923 referred to the heart as the "last frontier in surgery." His keen imagination and lively intellect would have hailed with enthusiasm the recent equally startling developments in the surgical correction of congenital malformations of the heart.

The most spectacular of these developments has been the application by Blalock[2, 3, 4] of Taussig's brilliant suggestion that in children, cyanosed and incapacitated by pulmonary stenosis or atresia, the blood flow through the lungs be augmented by an artificial, surgically created patent ductus. Taussig had noted that in cases of pulmonary stenosis the patent ductus was occasionally the only route by which unaerated blood reached the lungs and that over a period of years as the patent ductus slowly closed, the children gradually became more cyanotic and died. This possibility of gradual and finally fatal closure of an essential patent ductus had been suggested previously in a discussion of congenital intracardiac fistulas.[5]

To circumvent the pulmonary stenosis, Blalock developed the surgical technique of implanting a systemic artery into the pulmonary artery. In his first case he employed implantation of the left subclavian artery into the left pulmonary artery. Subsequently he implanted the left innominate artery of a right aortic arch into the left pulmonary artery, but his usual procedure has been to implant the right subclavian artery into the right pulmonary artery. The use of the innominate artery has been found dangerously hazardous because of the cerebral complications of hemiplegia or death incident to ligation of the common carotid artery, and is therefore avoided if at all possible.

Our experiences with these various procedures have been limited to thirty-four cases. In six instances for various reasons an anastomosis between the systemic and pulmonary vessels could not or was not performed. In one of these patients, instead of a pulmonic stenosis, a complete transposition of the pulmonary artery and aorta was encountered (Fig. 1), and the small 3-year-old child died almost immediately after the opening of the chest. A second child also died before any anastomosis could be effected. He was very blue and had had in his three years of life three embolic or thrombotic phenomena, including a cerebral accident producing a permanent left hemiplegia. In a third child, the application of the Blalock clamp for occlusion of the pulmonary artery caused cessation of cardiac contractions, and after several

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Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

such attempts, the heart ceased beating entirely. At autopsy it appeared that a barely patent and hidden ductus arteriosus lay in such a position as to be closed completely by the clamps on the pulmonary artery thus reducing the already greatly diminished blood flow to the lungs with resulting decreased aeration. In a fourth patient, 12 years of age, it was evident that the right subclavian artery could not be brought down for anatomosis to the right pulmonary artery without ligation and division of the carotid artery. Because of the high incidence of cerebral complications following ligation of the common carotid, the operative wound was closed, and it was proposed to open the left side of the chest at a later date for anastomosis of the left subclavian artery to the left

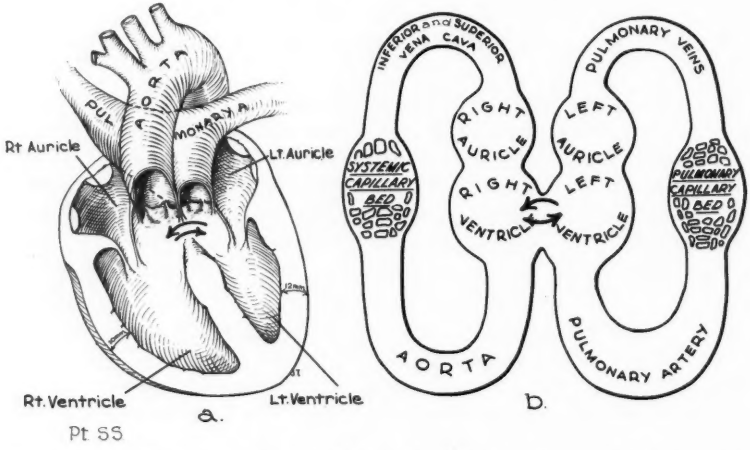

Fig. 1.—*a*, Complete transposition of pulmonary artery and aorta with an interventricular septal defect as the only communication between the systemic and pulmonic circuits.

b, Schema of circulation under these circumstances indicating that a to-and-fro flow of blood through the septal defect provided the only interchange of blood between the two systems. The increased pressure in the pulmonic circuit incident to anesthesia probably prevented the usual flow from the right heart into the left heart with a resulting greater flow of unaerated blood in the systemic circuit and death. The greater cyanosis, even under the administration of oxygen, and prompt death on opening the chest lends credence to this view.

pulmonary artery. However, the boy never recovered consciousness and died twelve hours later in marked and increased cyanosis. Apparently the mere administration of a general anesthetic had somehow altered the pressure relations between the systemic and pulmonary circulations (Fig. 2), causing a more than usual diversion through the interventricular septal defect of unaerated blood into the systemic circulation which was incompatible with life.

In a fifth child, 3 years of age, the exposed pulmonary artery was 1.8 cm. in diameter and pulsated vigorously. The water manometer was not quite high enough to measure accurately the pulmonary pressure, but it was estimated to be at least 900 mm. of water. The systemic arterial pressure at that moment was 98 mm. of mercury, and it was obvious that the pressures in the two systems were approximately equal. It seemed probable that we were dealing with a truncus deformity, and no anastomosis was performed. The child died a few hours later. Only a partial autopsy was permitted, but it disclosed that the

child had indeed a truncus arteriosus fed by both ventricles, thus accounting for the equal pressures in the pulmonic and systemic circulations. In a sixth child, at thoracotomy no pulmonary artery could be found on the right side, and the operation was abandoned. Two weeks later a left thoracotomy was performed, but again no pulmonary artery could be found. This 6-year-old boy is still living, no worse for the two unsuccessful explorations.

In twenty-seven cases the operation was completed with good to phenomenal improvement in twenty-four cases and death in three. The first death was in a 15-month-old infant whose operation was performed on Feb. 16, 1946. The right subclavian artery at the site of division and anastomosis

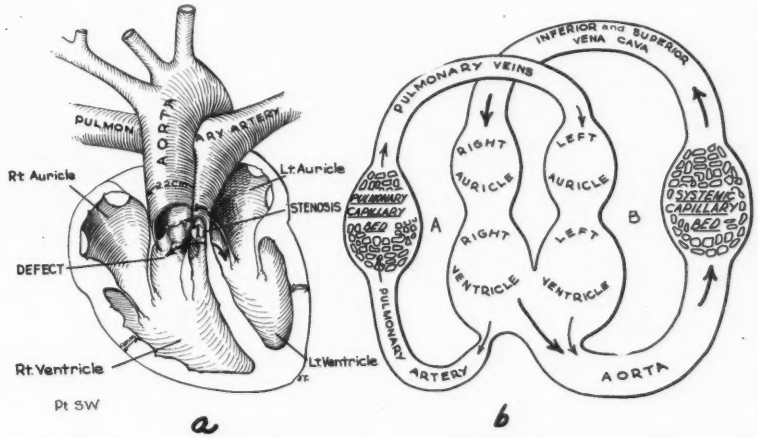

Fig. 2.—*a*, Marked pulmonic stenosis, interventricular septal defect, large overriding aorta in a 12-year-old boy who died twelve hours after simple thoracotomy.

b, Schema of circulation indicating that the increasing cyanosis and death after operation was probably explicable on the basis of an increased flow of unaerated blood into the aorta due to an increased pressure in the pulmonic circuit imposed by anesthesia.

to the pulmonary artery was only 3 mm. in diameter. At autopsy, thrombosis was found at the site of union, with partial angulation and obstruction of the right pulmonary artery, which had so reduced the flow of blood to the lungs that death occurred twenty-four hours after operation. This experience led to the use of the right common carotid artery in our next patient, only to have this patient develop a left hemiplegia from which she slowly recovered.

The second death occurred in a patient in whom the ligature on the distal end of the divided subclavian artery included, besides the vessel itself, some of the connective tissue fixed to the thoracic wall so that the ligature was anchored in place. The artery, which had been pulled down out of the tissues of the neck, was therefore held in place under considerable tension. The elasticity of its wall led to its being gradually pulled out of the anchored ligature, resulting in a fatal hemorrhage two hours after the operation. The obvious lesson is to ligate doubly or to ligate and transfix the distal artery, being careful also to avoid including in the ligature any surrounding fixed

tissues. An artery ligated under tension must be completely free of surrounding tissues so that it may retract into its normal position.

In the third patient, 19 years of age, who died with a hemoglobin concentration of 114 per cent, and a red cell count of 8.1 million, a very unsatisfactory anesthesia was administered, the patient's color being blue-black throughout the operation despite every effort to correct it. A good anastomosis between the left subclavian artery and the left pulmonary artery was affected, but the patient never recovered consciousness and died twenty-four hours after operation. An autopsy was not permitted, but cerebral anoxia was un-

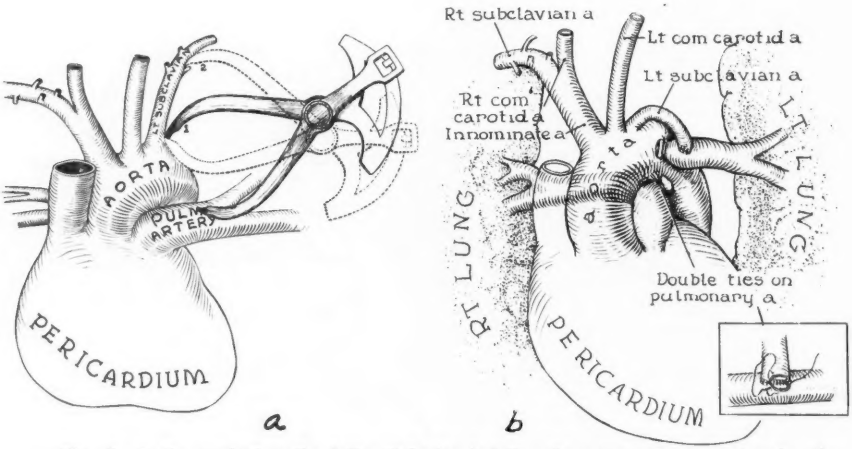

Fig. 3.—*a*, In employing the left subclavian-to-left pulmonary artery anastomsis, the distance between the base of the subclavian artery and the proposed site of anastomosis is measured by calipers, and an equal length of subclavian artery is mobilized for division at 2.

b, If the subclavian artery is sharply angulated at its orgin on opening the anastomosis, the pulmonary artery central to the anastomosis is doubly llighted and divided between ligatures, thus permitting the pulmonary artery to rise in the chest with correction of the angulation and an increased flow through the anastomsis.

doubtedly an important factor in this patient's death, as indicated by a left hemiplegia and a marked right-sided spasticity noted immediately after the operation.

In two feebleminded infants the innominate artery was sutured to the right pulmonary artery with excellent results, so far as physical activity and improvement in cyanosis were concerned. The mothers even believed there was some mental improvement.

Because of difficulties with the short right subclavian artery, and the distressing cerebral complications that may follow ligation and division of the right common carotid artery when the innominate is used the left subclavian artery was employed because of its greater length. The objection to its use has been made by Blalock, that in bringing it over the arch of the aorta the acuteness of the angle at its origin interferes with the flow of blood through it. This is a valid objection in some instances, but where such interference with blood flow occurs, it is quite obvious immediately

upon releasing the clamps and opening the anastomosis. Fortunately, it is readily corrected by complete division of the left pulmonary artery between the site of the anastomosis and the primary branching of the vessel (Fig. 3). That the patient can tolerate such division has already been demonstrated by its complete occlusion during the time in which the anastomosis was being made. Such complete division was found necessary, however, in only four of the twenty-three cases (Fig. 4). In a fifth case, a 9-year-old girl, the left pulmonary artery was only 7 mm. in diameter, too small for an end-to-side anastomosis. Accordingly, the artery was completely divided, and an end-to-

Fig. 4.—Marked muscular weakness in a cyanotic boy was completely corrected by a left subclavian to pulmonary artery anastomosis with division of the left pulmonary artery central to the end-to-side anastomosis (see Fig. 3). Following operation the patient *a* became quite as active as his twin *b*.

end anastomosis between the subclavian artery and the distal pulmonary artery was established with good functional results (Fig. 5).

In nineteen cases in which the left subclavian artery was sutured to the side of the left pulmonary artery without division of the pulmonary artery, good functional results have been obtained as shown by absence or improvement in cyanosis, by greater physical activity, and by absence of dyspnea. Some patients are said by their parents to be perfectly normal. The patients have ranged in age from 15 months to 26 years. One 18-year-old was practically incapacitated by dyspnea, necessitating squatting, even in the middle of a busy street, to recover his breath before he could go on. He is now able to hold a job (watch repairing), no longer squats, and has almost a normal color.

All patients but one were considered as belonging in the general group of tetralogy of Fallot. This one exception was a 5-year-old boy whose frank cyanosis did not appear until 3 months of age. His activity was greatly

limited, and cyanosis and dyspnea were aggravated greatly with the slightest exercise. He watched other children, rather than participating in their play. He had a loud systolic murmur at the base heard all over the precordium. The pulmonic second sound appeared loud and snapping. The circulation time from arm to lung was 6.5 seconds. The red cells numbered 7.8 million, and hemoglobin concentration was 142 per cent; blood pressure was systolic 110, diastolic 70. The electrocardiogram showed abnormal P waves and a left axis deviation. The roentgenogram showed a normally contoured heart. At operation the right subclavian artery, which was 8 mm. in diameter, was implanted into the right pulmonary artery 1.5 cm. in diameter. When the anastomosis was opened, the blood pressure fell to 104/56, and the pulse be-

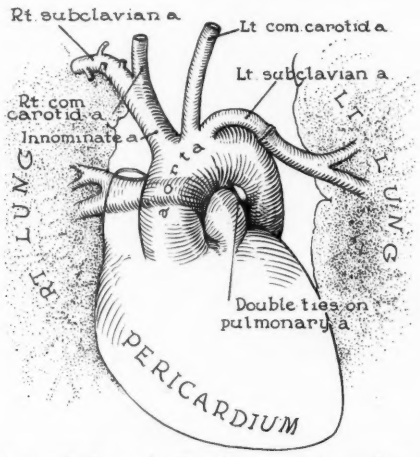

Fig. 5.—A pulmonary artery only 7 mm. in diameter may not permit an end-to-side anastomosis but will permit an end-to-end anastomosis between the subclavian and pulmonary arteries.

came accelerated from 110 to 120. Since operation, the cyanosis has practically disappeared, his physical activity is normal and as vigorous as that of his brother. The red cells now number 5.5 million and the hemoglobin concentration is 99 per cent. This patient was interpreted as having a tricuspid stenosis with an open foramen ovale, rather than a pulmonary stenosis with an open interventricular septum.

The use of angiograms* in selected cases has provided interesting and valuable help in arriving at more accurate diagnosis (Fig. 6).

Complications following operation have been few. One patient's course following operation on July 31, 1947, was characterized by a persistent chylothorax. Over 4,430 cm. of milky fluid were aspirated from the left side of the chest over a period of nine days. Intravenous alimentation with protein hydrolysate led to thrombosis of the right saphenous vein, resulting in a paradoxical embolism through the interventricular septal defect to the right cerebrum with

*These were made at the insistence of Dr. Ann Purdy, whose enthusiasm and aid in diagnosis, together with the cooperation of Dr. Harold Faber, both of the Department of Pediatrics, have made this work possible.

a left-sided hemiplegia on the seventeenth day following operation. Gradual recovery took place. On Nov. 17, 1947, he was reported as being able to walk alone but was still unable to use his arm.

Since this experience, lymphatics are carefully watched for, as the left subclavian artery is isolated from the mediastinum, and on one occasion a large vessel filled with clear fluid was observed to cross the operative area and was carefully avoided. In another instance turbid chylous fluid suddenly filled the operative area; the point of leakage in the mediastinum was sought, identified, and clipped with a silver clip, with no subsequent trouble.

In three patients, a chest filled with fluid led to the aspiration of 900 cm., 500 cm., and 1,200 c.c. of bloody fluid, respectively. All patients then

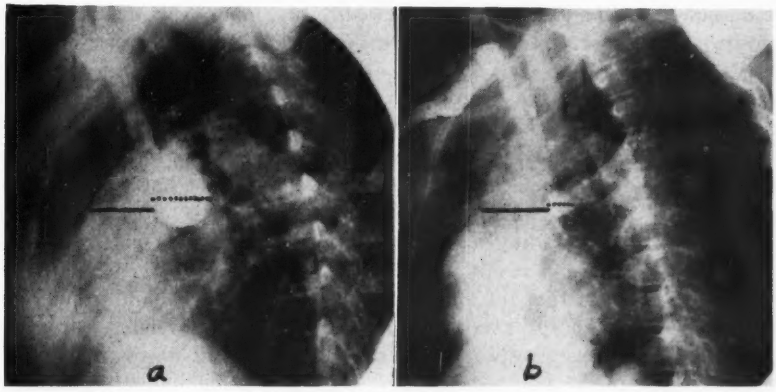

Fig. 6.—*a*, An angiogram in a 19-year-old girl, cyanotic since birth, reveals a normal sized pulmonary artery (dotted line) in relation to the aorta (unbroken line). This evidence was interpreted as supporting a diagnosis of Eisenmenger complex.

b, An angiolgram in a 12-year-old boy revealed a greatly narrowed pulmonary artery (dotted line) in relation to an oversized aorta (unbroken line). The early appearance of the dye in the aorta indicated the presence of a septal defect. Subsequent necropsy confirmed this relationship. (Same patient as shown in Fig. 2.)

had an uncomplicated recovery. In two of these patients pressure in the pulmonary artery had been ascertained by introducing a needle for manometric readings. It was surmised but not proved that the bleeding came from this puncture wound.

Pulmonary arterial pressures at operation have ranged from 175 to 370 mm. of water. Although no arbitrary level can be set, Blalock has stated that the operation should not be done when the pulmonary pressure exceeds 300 mm. of water. In the 4-year-old child in whom the pulmonary pressure was 370 mm. of water, the systemic arterial pressure at that same moment was 110 mm. of mercury. It was reasoned that a flow of blood would therefore occur from the systemic to the pulmonary circulations, and accordingly the left subclavian artery was implanted into the left pulmonary artery with excellent improvement in cyanosis and physical activity. The mother writes that he plays normally, whereas formerly he was content simply to observe his playmates.

Improvement following operation is disclosed not only by marked improvement in appearance and physical activity, but also by marked change in the character of the blood; in one instance the number of red cells dropped from 11.4 million to 6.9 million, and the hemoglobin concentration from 188 per cent to 103 per cent. In another case, the red cells fell in number from 7.8 million to 5.5 million and the hemoglobin concentration dropped from 142 per cent to 99 per cent.

Various operative approaches have been used. At first, Blalock's anterior incision through the third interspace was employed. This has been replaced in girls by a left anterolateral approach through the third interspace to avoid conspicuously visible anterior scars and possible injury to breast tissue. In boys an anterior approach through the second interspace is now used, with division of the second and third costal cartilages. This provides an excellent exposure of the left hilum and more ready access to the left subclavian artery.

In performing the anastomosis, a longitudinal rather than a transverse incision is now made in the pulmonary artery, and the suture employed is a continuous adventitia-to-adventitia approximation in the posterior half, and a continuous intima-to-intima approximation in the anterior half of the circular anastomosis. An effort is made to interrupt completely the circular silk suture at two points, thus avoiding a complete circular suture of nonabsorbable silk which might conceivably result in a fibrous, nondistensible rim to the circular opening. It is hoped that the anastomosis will dilate as the child grows and as more blood is needed for aeration. In peripheral arteriovenous fistulas such dilatation of the anastomosis itself does occur, its extent depending upon the original size of the fistula, its duration, and its location in the vascular tree. Moderate and gradual increase in the size of the anastomosis in the Blalock procedure is probably desirable and necessary if the volume of blood passing through the anastomosis for aeration in the lungs is to increase commensurate with the increase in size and weight of the child and with the increase in total volume of blood. Unless such an increase in blood flowing through the anastomosis takes place, it seems probable that the patient will again show signs of inadequate oxygenation as bodily growth takes place.

It must be recognized that the establishment of this artificial patent ductus carries with it the same hazards as those associated inevitably with the congenitally patent ductus, namely, cardiac failure, bacterial endocarditis, and endarteritis. The latter may be controlled by antibiotic therapy, the former may be without remedy. Inasmuch as the resistance to the flow of blood through the pulmonary circuit will always be less than the resistance to the flow through the systemic capillary bed, there will be a constant tendency for more blood to be diverted through the anastomosis and therefore through the lungs and the left heart, which may lead ultimately to an overburdening of this half of the heart. It seems quite probable, therefore, that these patients will eventually succumb to the same difficulties which are associated with a patent ductus, and that their life expectancy is no greater than that of the patent ductus, perhaps even less, because of the associated anomalies of a

patent interventricular septal defect, and right-sided cardiac hypertrophy. In the meantime, however, the greatly improved physical status of these unfortunate little patients is a great boon to distraught and solicitous parents.

SUMMARY

Thirty-eight patients with cyanosis have been operated upon.

In seven, thoracotomy without anastomosis was performed with five deaths. These fatalities are explained on an increased deflection of unaerated blood into the systemic circulation through the interventricular septal defect, due to the increased peripheral resistance in the pulmonary vascular bed incident to anesthesia and to the opening of the chest.

In twenty-nine patients, a systemic artery was joined to the pulmonary artery as proposed by Blalock: in two, the innominate artery was joined to the pulmonary artery with excellent results; in one, the right common carotid artery was united with the right pulmonary artery, followed by a temporary left hemiplegia; in three, the right subclavian artery was joined to the right pulmonary artery with one death (in a patient 19 months old) and with two excellent results.

In twenty-three patients, the left subclavian artery was joined to the left pulmonary artery with two deaths, and twenty-one good to excellent results. In four patients the left pulmonary artery was completely divided proximal to the anastomosis because of sharp angulation at the origin of the subclavian artery which prevented a free flow of blood through the anastomosis. Following such division, the distal pulmonary artery rose in position in the chest, the angulation was corrected, and a good flow through the anastomosis occurred.

It is suggested that the left subclavian artery is preferable to the right because of its greater length, and if angulation at its origin occurs following its anastomosis, such angulation can be corrected by complete division of the pulmonary artery proximal to the end-to-side anastomosis.

In two patients, each 9 months old, the Potts procedure of direct anastomosis between pulmonary artery and arch of aorta was performed with one excellent result and one death.

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DISCUSSION

DR. F. D. DODRILL, Detroit.—I wish to speak about a small group of patients in whom the left subclavian artery was used for the anastomosis to the left pulmonary artery. During the last twelve operations for tetralogy of Fallot, I have used the left-sided approach, a left aortic arch being present in all cases, hoping to use the left subclavian artery. An anastomosis was carried out in eleven of these patients. In ten of the patients it was quite satisfactory, there being no angulation of the subclavian at its origin from the aorta. However, in one patient there was angulation of the subclavian artery as it arose from the aorta. Not knowing of Dr. Holman's technique, I freed up the outer portion of the aortic arch and thought that the angulation was definitely less. It is now six months since the operation, and there is good clinical response. In the one patient in whom the subclavian artery was not used, a Potts aortic-pulmonary artery procedure was carried out with good results. The youngest patient in this group was 15 months old, the oldest 22 years. In older children and adults the left subclavian artery is longer and, therefore, there is a better chance that it will reach to the pulmonary artery.

In this group in which left-sided approaches were used there was one death; a 22-year-old man died of embolism arising in the lower extremity. At autopsy there were emboli in the lungs, brain, and kidney. The anastomosis was fully patent without evidence of thrombi at the suture line. The remainder of the patients have had a good clinical response. The oxygen saturation has increased, the red blood count has returned to normal or near normal, and the tolerance to exercise has markedly increased. Previous to this time there were twenty-six operations carried out on the right side, using the right subclavian artery, but in a few of the earlier cases, the carotid artery was used. There were five deaths in this group. In two instances, however, death occurred as the chest incision was being made and before the vessels were isolated.

If the subclavian artery and a posterior approach are being used, I believe it is important not to carry the incision too high on the chest wall, as some of the collateral circulation to the arm might be sacrificed.

DR. HOWARD T. BARKLEY, Houston, Texas.—We have had only a small series of cases, but I wish to add a few words to the experiences of the essayist and discussers.

We have been impressed by the difficulty in making a definite diagnosis in these cases preoperatively. Perhaps with the use of angiograms, which we have not had available up to the present time, we can expect further aid in definite diagnosis; however, it is our feeling that in most of these cyanotic patients exploration should be carried out on the chance that some situation will be found that can be helped by anastomosis. We found one such case with a common truncus arteriosus in which the pulmonary artery was exceptionally small. We feel that this patient would have been benefited by the anastomosis of a larger vessel to the pulmonary artery, but unfortunately the patient died on the operating table. This is the only death we have had at operation. In dissecting out the pulmonary artery on the left side, we had completely occluded the blood flow to the lungs by tension at the bifurcation of the main pulmonary trunk, thus producing a complete anoxemia. This was shown to be true at autopsy; therefore, we advise great caution in dissection of the vessels in the mediastinum until the true situation can be seen.

We have preferred the posterolateral approach because of the additional working space and better visualization, and we prefer to the left-sided approach if there is no apparent contraindication. In one of our early cases, we used the right-sided approach because of a left aortic arch, expecting to use the right innominate or subclavian in a very small child. We were unable to find a pulmonary artery on the right side and had to withdraw; therefore, we have felt we would always have a better opportunity using the left side in exposing the base of the heart and finding even a small pulmonary artery, if there is one present, and perhaps being able to make some type of anastomosis rather than having to admit defeat.

DR. CLARENCE CRAFOORD, Stockholm.—I would like to make a few brief comments and show two slides. We now always try to get an anatomic diagnosis through angio-

cardiograms before operation. I know that the master in this field considers cardiograms not to be necessary; by information from good cardioangiograms we get, preoperatively, information as to how the pulmonary artery, the aorta, and the big vessels leaving the arch are localized and information on the size of the pulmonary artery and its branches. We perform the cardioangiograms in children most often under general anesthesia, either with injection in the cubital vein or, if this method does not give satisfactory results, through the heart catheter when all information at catheterization is obtained. These exact data are of importance for the planning of the operation. The following slides will demonstrate what I have now said.

(Slides.) Where there is difficulty in bridging over the gap between the subclavian artery and the pulmonary artery when performing the end-to-side anastomoses between these vessels, I have found it most helpful to divide the pulmonary ligament up to the lower pulmonary vein, which permits the whole hilum of the lung to be considerably elevated in a cranial direction, thus facilitating the anastomoses without tension.

DR. JULIAN JOHNSON, Philadelphia.—We have encountered two patients in whom the subclavian artery was so short that it seemed to us that it would be impossible to use it to do the Blalock operation. On the basis of experimental work in the laboratory, we felt that we could safely do a vein graft to increase the length of the subclavian artery, so that it could be anastomosed to the pulmonary artery. In both instances, we took a section of the superficial femoral vein and used it as a graft between the end of the right subclavian artery and the right pulmonary artery. Both patients obviously had functioning anastomoses with a left or right shunt. Unfortunately, one of these patients died from a pulmonary embolus, apparently having arisen at the site where the vein was taken in the superficial femoral area and having lodged in the pulmonary artery at the point of stenosis. The other patient, however, has done exceedingly well and has a loud continuous murmur. It would seem to us that when one encounters the situation of an inadequate length to the systemic vessel, that if other maneuvers are not adequate, one can safely use a vein graft to fill the gap to the pulmonary artery.

DR. ALFRED BLALOCK, Baltimore.—I want to congratulate Dr. Holman on this excellent presentation. The suggestions which he has made regarding points in operative technique are good. In this connection I would like to caution that the clamp which we devised for occluding the pulmonary artery should not be used on systemic arteries. If the clamp is applied tightly, it will crush the intima of a systemic artery.

Dr. Crafoord, I wish we felt as confident of the diagnosis in all cases as you seem to think we do. Even so expert a diagnostician as Dr. Taussig is not always certain as to the exact nature of the malformation. As an example, I have performed anastomoses on at least five patients who had a transposition of the great vessels under the assumption that we were dealing with pulmonic stenosis.

It was my impression prior to 1947 that an artificial ductus should not be created in the treatment of pulmonic stenosis if the pressure in the pulmonary artery is greater than 350 mm. of water. Subsequent experience has shown that this impression is wrong. Several months ago an artificial ductus was created in a patient in whom the pressure in the pulmonary artery was 530 mm. of water. The pulmonary artery was of good size, and the pulsations were strong. This patient has improved markedly following the operation.

In regard to the site of the incision we still use the anterior approach but feel that there is no strong objection to the posterolateral approach. We now make the incision into the chest through the second interspace instead of the third. I would like to show a few lantern slides of drawings which demonstrate the various operations which may be used. These show an end-to-side anastomosis between the subclavian branch of the innominate and the right pulmonary artery, and end-to-side anastomosis between the subclavian branch of the aorta and the pulmonary artery, and end-to-end anastomosis between the subclavian artery and the pulmonary artery, and a side-to-side anastomosis between the aorta and the pulmonary artery. I would like to emphasize that a good end-to-end anastomosis is preferable to a fair end-to-side one.

In conclusion, I would like to express an opinion as to the choice of operative procedure under various conditions. If the patient's aorta descends on the right instead of the left, which occurs in about 22 per cent of the cases, I think that the incision should always be made on the left and an anastomosis performed between the subclavian branch of the innominate and the side of the left pulmonary artery. When the innominate is on the left, there is rarely any difficulty in approximating its subclavian branch to the pulmonary artery. Approximately 155 of our 650 patients have had an aorta which descended on the right. When the aorta descends on the left, we vary the approach somewhat according to the age and size of the patient. In patients between the ages of 2 and 12 years, and these constitute the majority, the incision is made on the right, and the end of the subclavian branch of the innominate is usually anastomosed to the side of the right pulmonary artery. In patients who are more than 12 years of age, we usually make the incision on the left and use the subclavian branch of the aorta. This is the procedure about which Dr. Holman has spoken. The left pulmonary artery is at a slightly higher level than the right, and this aids in the approximation. In patients under 2 years of age, the choice varies. If we think that the subclavian artery is large, normally we make the incision on the right and use the subclavian branch of the innominate. If it is thought that the subclavian artery is quite small, we make the incision on the left. If the subclavian is found to be large enough, we anastomose it to the left pulmonary artery. If the subclavian appears too small, we perform a side-to-side anastomosis between the aorta and the left pulmonary artery as developed by Dr. Potts. We do not advise operation on patients under the age of 2 years unless they are doing very poorly. The important point to remember regarding the operative procedure is that one should be aware of the various methods that may be used since there is no single procedure that can be applied satisfactorily to all patients. Most of the unfortunate experiences arise as the result of a poorly developed or absent pulmonary artery. There is always a systemic artery which can be utilized.

DR. HOLMAN.—I want to thank the master for giving us the final word on this subject. We, too, have found that we could perform anastomosis with pressures in the pulmonary artery as high as 420; we have not gone higher than that.

DEVELOPMENT OF ANASTOMOSIS BETWEEN THE CORONARY VESSELS AND A TRANSPLANTED INTERNAL MAMMARY ARTERY

ARTHUR M. VINEBERG, M.Sc., PH.D., M.D.C.M., MONTREAL, QUEBEC

DURING the past fifteen years many attempts have been made to augment impaired coronary circulation. Three main procedures have been described. The first of these is dependent upon the grafting of a vascular structure to the ventricular wall such as the pectoral muscle graft of Beck in 1935 or the great omental graft of O'Shaughnessy in 1937.

The second procedure employs the parietal pericardium as a source of fresh blood. Talc powder or asbestos fibers are introduced into the pericardial sac. The reaction caused by the foreign body particle stimulates the formation of granulation tissue between the visceral and parietal pericardium.

The third procedure, described by Fauteux in 1940, depends upon ligation of the vena magna cordis to increase ventricular circulation.

All three procedures have been carried out upon human cases of coronary artery disease. The first two methods bring a fresh supply of arterial blood to the ventriclue through a layer of granulation tissue. In time the granulation tissue contracts into scar with resultant choking off of the newly formed blood vessels. The third procedure relies upon venous occlusion to open anastomoses between the partially thrombosed coronary arteries, arterial luminal vessels, and arterial sinusoidal vessels.

The basic objective in this work has been to supply a fresh source of arterial blood to the heart muscles. In order to do this, the left internal mammary artery has been partially removed from its normal position on the chest wall and implanted into the myocardium of the left ventricle. In this way we thought that a communication might be developed between the implanted internal mammary artery and the left coronary circulation.

METHOD

Basically, the method consists of freeing the left internal mammary artery from the chest wall for variable distances. All intercostal branches are tied and severed over the free part of the artery. The artery after being doubly ligated is cut across at its distal end. The proximal free end of the artery is drawn through a previously prepared tunnel in the anterior wall of the left ventricle where it is fixed. All operations were carried out under aseptic conditions and the animals were kept from six weeks to thirteen months. At the end of this time they were either sacrificed by bleeding or the anterior descending branch of the left coronary artery was ligated at its origin. The circumflex branch of the left coronary artery was ligated in those animals surviving an anterior descending branch interruption. After death the internal

This work was assisted by a grant-in-aid from the Division of Medical Research of the National Research Council.

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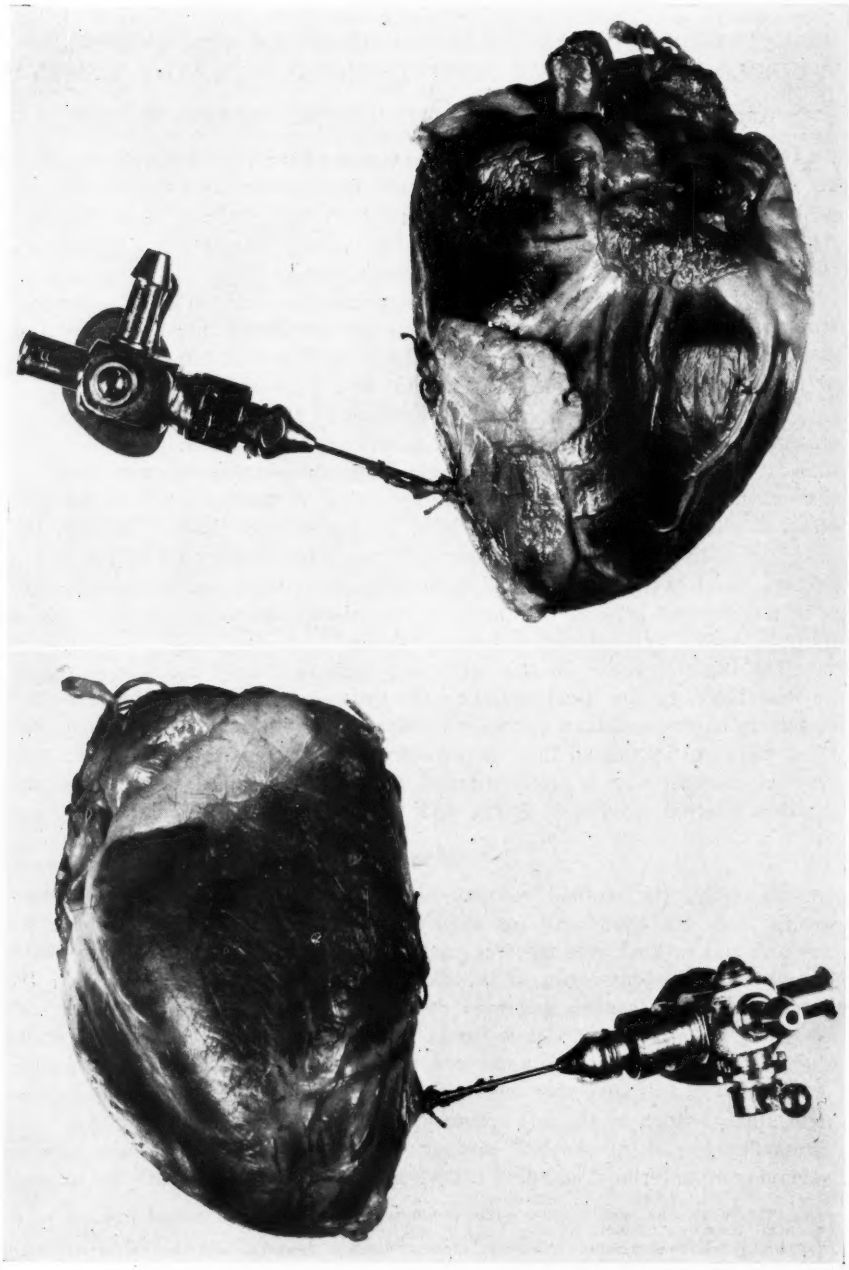
Read at the Twenty-eighth Annual Meeting of The American Association for Thoracic Surgery, Quebec, Canada, May 31, June 1, 2, 1948.

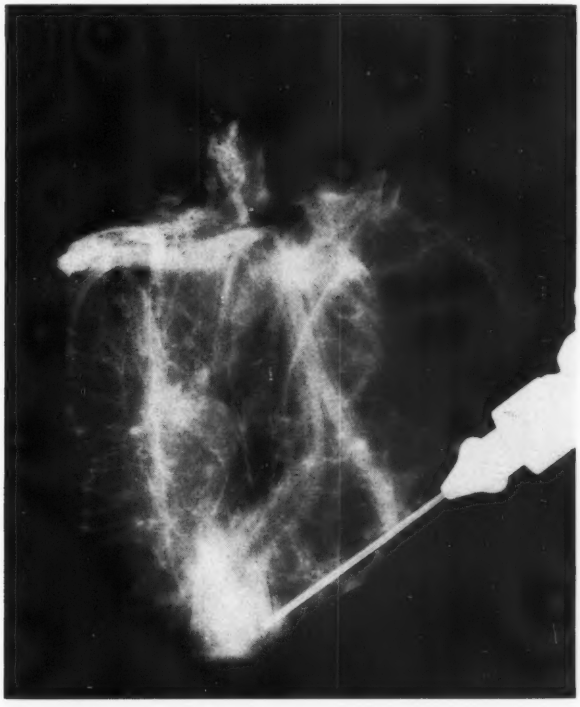
A.



B.

Fig. 1, *A* and *B*.—These show the heart of a dog killed four months after the left internal mammary artery had been implanted in the wall of the left ventricle. The internal mammary artery had been injected with pink Schlesinger's solution. Note that the vessels of the left ventricle only have been filled by the injection fluid.

mammary artery was cannulated and its distal end injected proximal to the heart. The Gross or Schlesinger technique of injection was employed. Certain specimens were injected with a plastic solution and a cast was made of the arterial lumina.



C.

Fig. 1, *C.*—X-ray of the left coronary tree which has been filled by injecting the left internal mammary artery four months after it was implanted into the left ventricular wall.

PROOF OF ANASTOMOSIS

The presence of anastomosis between a transplanted left internal mammary artery and the left coronary circulation has been studied by means of

(1) x-ray examination of injected specimens,
(2) serial sections taken through the site of anastomosis, and
(3) the making of a plastic cast of the arteries involved to show the nature of anastomosis.

The photographs shown in Fig. 1, *A*, *B*, *C*, *D*, *E*, and *F* represent studies made on Dog A5 in which the internal mammary artery was transplanted into the left ventricle on Feb. 4, 1946. The dog was killed on June 14, 1946, approximately four months later. The photographs shown in Fig. 1, *A*, and *B* are of the same heart after it has been injected by a pink Schlesinger solution. It should be noted that the vessels of the left ventricle only have been filled.

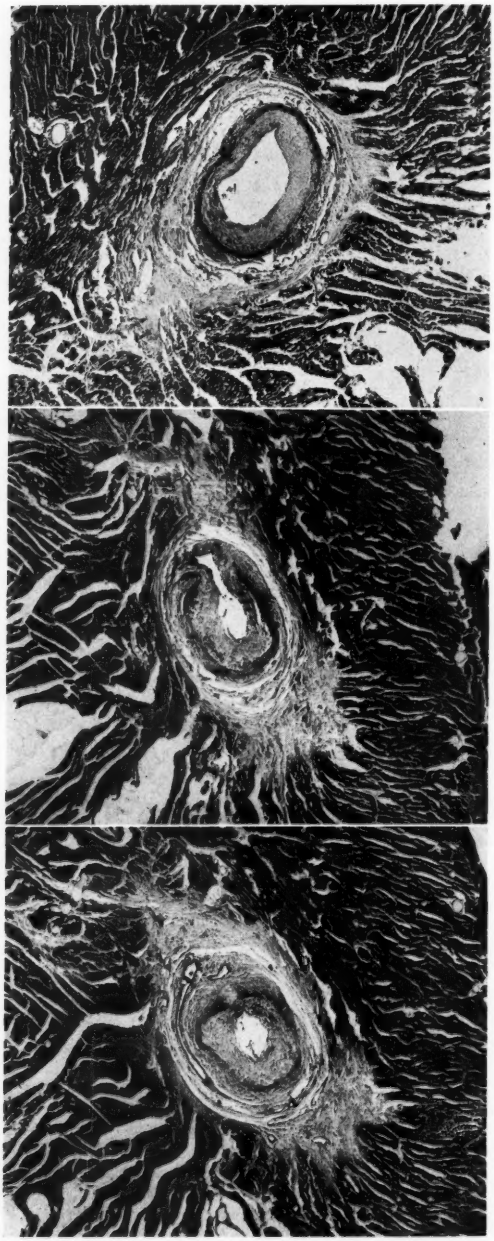


Fig. 1, *D*, *E*, and *F*.—The transplanted internal mammary artery lying within the muscle of the myocardium at the end of four months. Note that the only change to be observed is a thickening of the intima. *D*, It will be noted that in the upper portion of the section there is an indentation which is the beginning of a branch. *E* shows the internal mammary artery with the branch well developed. *F* shows a branch of the internal mammary artery lying alongside of the parent vessel.

The x-ray studies (Fig. 1, *C*) showed that the left coronary artery tree was filled by injecting the left internal mammary artery four months after its implantation into the left ventricular wall.

Serial sections (Fig. 1, *D*, *E*, and *F*) taken through the site of the internal mammary artery implantation showed:

1. In the upper portion of the section there is an indentation which is the beginning of a branch.

2. The internal mammary artery with branch well-developed.

3. A branch of internal mammary artery lying alongside of the parent vessel.

Photographs shown in Fig. 2, *A*, *B*, *C*, *D*, *E*, and *F* represent studies made on Dog A6 in which the internal mammary artery was transplanted into the left ventricular wall on Feb. 11, 1946; the dog was killed March 25, 1947, approximately thirteen months later.

Fig. 2, *A*, and *B* shows the filling of the left coronary circulation after the internal mammary artery was injected with Schlesinger solution (blue). Serial sections (Fig. 1, *C*, *D*, *E*, *F*, and *G*) show an indentation developing in the wall of the artery (internal mammary) lying within the ventricular myocardium. This indentation gradually develops, becomes larger, and finally separates into an independent branch which is seen lying alongside of the parent vessel.

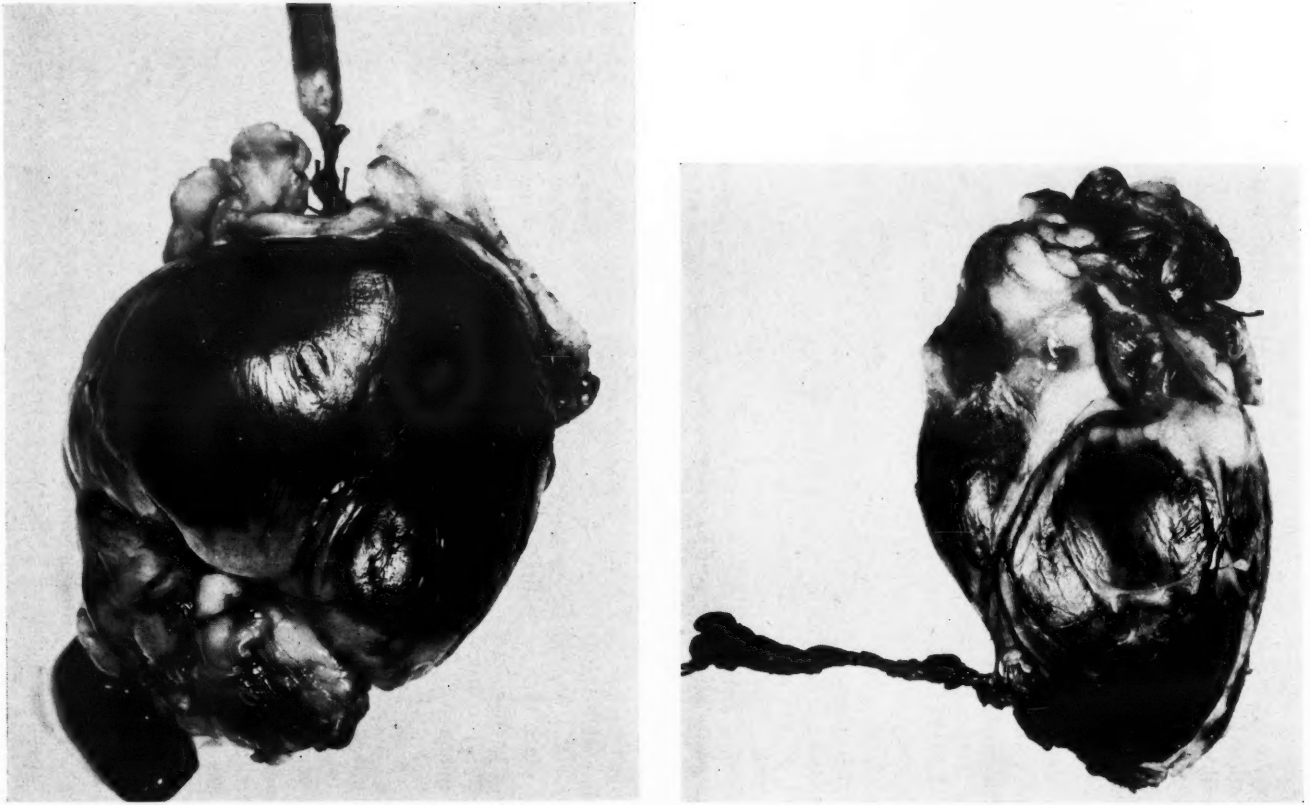
The photograph shown in Fig. 3 represents a plastic cast which was made of the internal mammary artery and the left coronary circulation on Dog A10. This animal had a left internal mammary artery transplanted into the left ventricular wall on June 21, 1946. The anterior descending branch of the left coronary vessel was ligated on April 12, 1947. The dog was killed on May 12, 1947, at which time a plastic cast was made of the internal mammary artery. It should be noted that the intercostals which had been tied off by cotton sutures failed to branch, whereas that part of the artery lying within the heart showed numerous new branches.

RESULTS (TABLE 1)

Results are divided into four main groups. Division depends upon the method used to prepare the iternal mammary artery for transplantation into the left ventricular wall.

Group 1.—The internal mammary artery was freed from the sixth intercostal branch to the subclavian artery. By this means a very long piece of artery was obtained which could be implanted into the left ventricular wall without angulation. A total of thirty-nine dogs was operated upon of which fourteen survived more than one month. In this group there was not a single case of arterial anastomosis.

Group 2.—The internal mammary artery and vein were freed together from the sixth intercostal to the fourth inclusive. The total number of animals operated upon was eighteen. Those that suvived were eleven in number. There was one (9 per cent) anastomosis.



A. *B.*

Fig. 2, *A* and *B*.—Cannula in the left internal mammary artery thirteen months after the artery had been implanted in the wall of the left ventricle. Note that the injection fluid has flowed down the artery into the left coronary circulation and out into the pulmonary vein. It has also flowed into the chest wall. Examination of *B* demonstrates that only the left coronary vessels were injected to the complete exclusion of the vessels of the right ventricle.

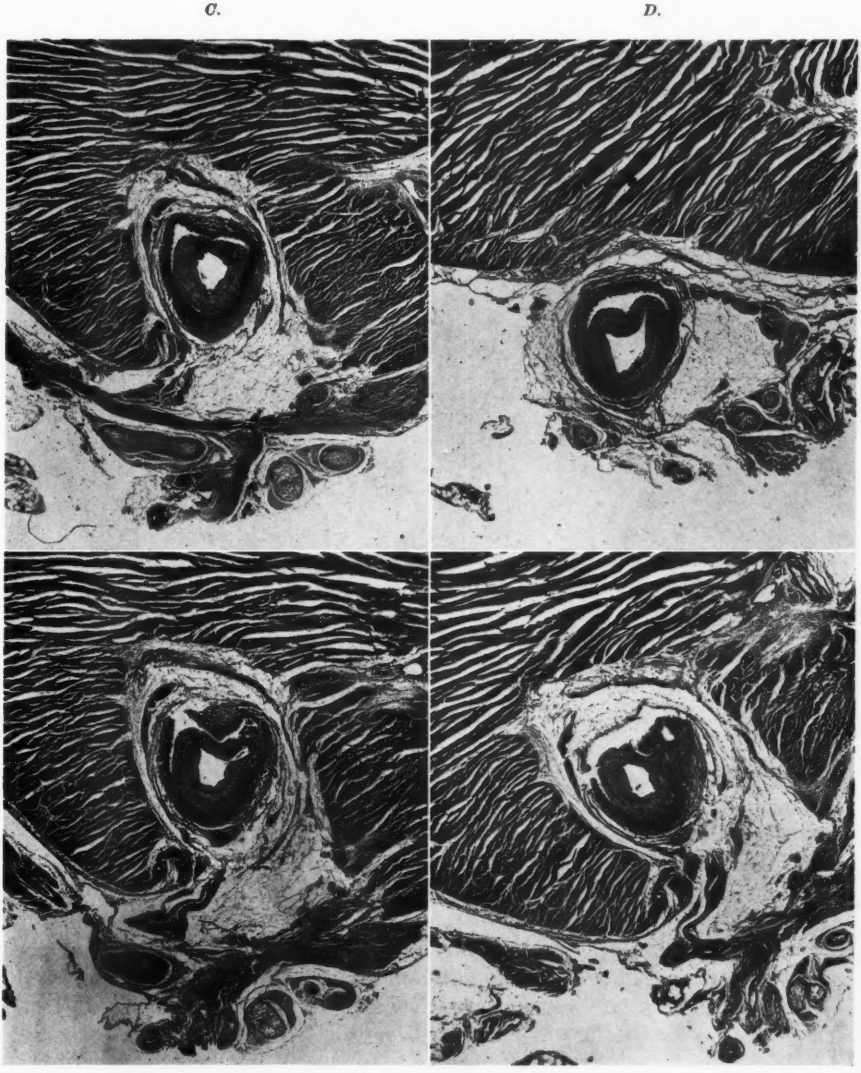


Fig. 2, *C*, *D*, *E*, and *F*.—Serial sections taken through the internal mammary artery, at the site of its implantation into the left ventricular wall (Dog A-6). In *C* there is an indentation in the arterial wall, which gradually develops into a second artery as shown in *C*, *D*, *E*, and *F*. The new artery is seen lying alongside of the parent vessel.

Group 3.—The internal mammary artery was freed from the sixth to the second intercostal arteries. The total number of dogs operated upon was thirty-one, of which fourteen survived. There were three (20 per cent) animals in which there was a proved anastomosis and one in which the anastomosis was suspected.

Group 4.—The artery was freed between the sixth and fourth intercostal vessels. In this group there were sixteen animals of which nine survived more than one month, and there was a proved anastomosis in four (44 per cent) and suspected anastomosis in two animals.

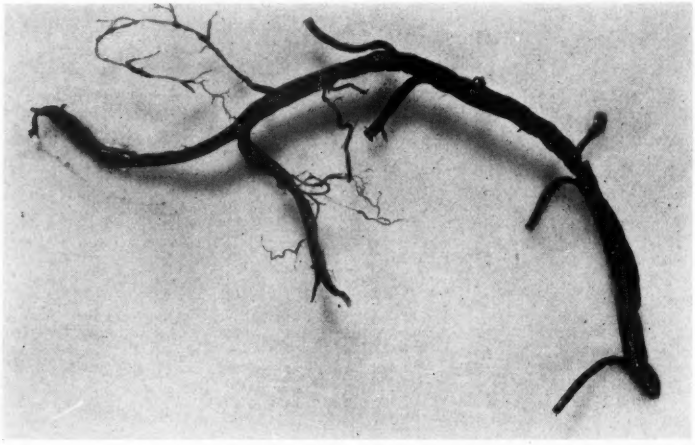


Fig. 3.—A cast has been made by injecting the internal mammary artery with a plastic substance ten and one-half months after implantation into the left ventricular wall. The small sharply cut off branches to the right are intercostals tied with cotton. The fine arborizations to the left have developed within the heart muscle.

When anastomosis occurs between the transplanted left internal mammary artery and the left ventricular vessels, there appears to be no connection between this anastomosis and the coronary vessels of the right ventricle. There is evidence to show that when this anastomosis is present it does not protect against a circumflex branch ligation. On the other hand, there is evidence to suggest that the new vessels will protect the left ventricle when the anterior descending branch of the left coronary artery is ligated at its origin. A total of twenty-nine dogs was subjected to ligation of the anterior descending branch of the left coronary artery at its origin following transplantation of the internal mammary artery into the left ventricular wall. Of these, twenty-four died within twenty-four hours of ligation and there were five which survived. It should be noted that of the five survivors there were three in which there was no anastomosis. All three showed massive ventricular wall infarction. Two of these showed residual branches of the anterior descending artery which were not included in the ligature. One survived in spite of infarction and a ligature properly placed to include all branches. Two of the five showed definite anastomosis with the left coronary circulation and no evidence of a ventricular wall infarction.

TABLE I. RESULT OF TRANSPLANTATION OF INTERNAL MAMMARY ARTERY INTO WALL OF LEFT VENTRICLE

DISTANCE OVER WHICH INTERNAL MAMMARY ARTERY FREED	NO. OF DOGS	LIVED MORE THAN ONE MONTH	PROVED ANASTOMOSIS	SUSPECTED ANASTOMOSIS	PATENCY OF ARTERY						REVASCULARIZATION OF CHEST WALL OR PERICARDIUM
					MORE THAN ONE MONTH			LESS THAN ONE MONTH			
					THROUGHOUT	TO SCAR	BLOCKED	THROUGHOUT	TO SCAR	BLOCKED	
Group 1 6th intercostal to subclavian	39	14	0	0	1	5	4	13	2	3	10
Group 2 6th to 3rd intercostal with vein	18	11	1 (9%)	0	1	9	0	1	0	0	Not recorded
Group 3 6th to 2nd intercostal	31	15	3 (20%)	1	3	7	0	5	1	2	9
Group 4 6th to 4th intercostal	16	9	4 (44%)	2	1	3	0	3	0	0	4
Total	104	49	8 (16%)	3 (6%)	6 (12%)	24 (49%)	4 (8%)	22	3	5	

DISCUSSION

Certain facts have been established which are difficult to explain. It is not clear what the mechanism is which permits an artery with one end closed to remain patent for many weeks and even months. Nor is there an explanation for the tendency of a transplanted internal mammary artery to form new branches when placed in a new vascular bed. In practically all instances when the artery was occluded by scar or a thrombus within the heart, new branches were sent out to the pericardium and chest wall. In many instances the artery reformed a completely new set of intercostal vessels. There is evidence that angulation of the artery plays an important part in its occlusion and that traumatization of the artery results in thrombus formation which might be diminished by the use of anticoagulants.

Certain technical difficulties which have been encountered in the dog should not be met with in man. The proximity of the human heart to the chest wall, and thus to the internal mammary artery, makes the possibility of angulation less likely. The mediastinum is nonexistent in the dog which prevents an extrapleural approach to the artery which will be possible in man. The extrapleural approach should materially diminish scarring of the artery. The accumulation of intrapleural fluid is practically impossible to control in dogs. Control of pleural fluid and the use of anticoagulants present no great problem in the human being. In the dog the internal mammary artery has been transplanted into a myocardium where there was a good arterial pressure. In man suffering from coronary artery disease, this would not be true. For these reasons it is thought that transplantation of the internal mammary artery into the wall of the left ventricle, in human cases of coronary artery disease, would have a better chance of forming a new vascular bed in man than in the dog.

CONCLUSION

The left internal mammary artery, after implantation into the left ventricular wall, formed a communication with the circulation of the left coronary vessels.

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DISCUSSION

DR. SAMUEL ALCOTT THOMPSON, New York.—For approximately nine years we have been using a form of surgical treatment for patients having coronary disease and angina. We consider this a form of rehabilitation rather than cure of the patient. We use U.S.P. talcum powder inside the pericardial sac, which produces two results: establishment of pericardial adhesions and myocardial hyperemia. The myocardial hyperemia is profuse and prolonged. It is likely that a form of talcum powder granuloma is produced which lasts for many years, and this is characterized by increased vascularity and hyperemia. This is a reversal of the myocardial ischemia which occurs in coronary artery disease.

In a series of thirty-six seriously handicapped patients, six have died. Of the remaining patients, 85 per cent are improved, and these results are classified as good or excellent. In four patients the results were poor. Eight patients have been completely relieved of

their angina and consider themselves to be practically normal. The criteria of rehabilitation are first, relief of anginal pain; second, an increase in the exercise tolerance; third, the ability to take care of their daily needs so that moving about and traveling is possible, and fourth, a return to their former, or at least some gainful, occupation.

The need for rehabilitation of such cases becomes apparent from a study of the vital statistics. In 1945, the total death rate in the United States was approximately one and one-half million; almost 30 per cent of these were cardiac deaths, more than 400,000. Of the cardiac deaths, over 130,000, or approximately 10 per cent of the total death rate, were deaths from coronary disease. It is likely that a large percentage of this group was incapacitated to some degree before death.

This is a simple method of treating such cases. We do not believe it leads to scar tissue or constrictive pericarditis. The patients are checked for this at intervals by measuring the venous pressure, and in none of the patients has the venous pressure gone above its preoperative level. If such a condition were going to develop it seems that it would have developed in nine years.

DR. ALFRED BLALOCK, Baltimore.—I do not think that I would have the temerity to discuss this paper if Dr. Claude Beck, who has performed such monumental work on the blood supply of the heart, were here. As some of you know, Dr. Beck and his associates have reported recently on experiments in which one of the systemic arteries was anastomosed to the coronary sinus. Dr. T. N. P. Johns and I performed similar experiments, as have others, and I would like to speak briefly of these. In some of these experiments the proximal end of the divided subclavian artery was anastomosed to the side of the coronary sinus and in other experiments the proximal end of the carotid was anastomosed to the divided end of the coronary sinus. The purpose, of course, was to try to cause a retrograde flow. In the experiments in which an end-to-side anastomosis was performed, most of the flow of blood went directly into the right auricle. In the experiments in which an end-to-end anastomosis was performed the immediate mortality was quite high and the incidence of thrombosis of the anastomotic site in those that survived was quite high. I wish to show lantern slides of the autopsy findings in three experiments in which survival occurred and the anastomosis remained patent. The coronary sinus becomes markedly dilated and thickened. We are unable to say whether retrograde flow through the capillaries occurred.

The point I wish to make is that this is a highly dangerous operation even in the experimental animal and that the incidence of thrombosis in the experimental animal is quite high. When one considers that the average length of life in patients who survive the first attack of coronary occlusion is approximately five years, I think that one should be very reluctant to use such an operative procedure. Only yesterday Dr. Churchill and I took a long walk with a surgical colleague who had had his first episode of coronary occlusion twenty years ago and there have been repeated episodes since then. I think that both Dr. Churchill and I were more exhausted at the end of this walk than was our surgical colleague. I do feel, however, that it is very important to continue to carry out experiments on attempts to increase the blood supply of the heart.

DR. F. R. MAUTZ, Cleveland.—I have enjoyed Dr. Vineberg's paper very much, and I cannot refrain from making a few remarks concerning my own experiences while working with Dr. Beck on the original idea of producing new arterial anastomoses. It has now been well established that new arterial anastomoses between a coronary artery and an extracoronary artery can be produced by man. When first formed these vessels are small, of 100 micra or less in diameter, and therefore they cannot carry very much blood. However, if there is slowly progressive occlusion of the coronary artery, then these collaterals increase in size. However, the first channels to enlarge are the pre-existing anastomoses between the adjacent coronary arteries. We have seen instances in the experimental animals where these vessels have increased one hundred times in cross section. When both coronary arteries are slowly and progressively obliterated it is then that the extracoronary anastomoses becomes more significant. The heart can run on perhaps less than

one-third of the total available coronary arterial capacity, of course with some restriction of activity. So we might say that the arterial collaterals prefer to follow the shortest detour around an obstruction, and I believe there is much to be learned about the factors that cause these arterial collaterals to enlarge once they are formed. We have observed one fact that may offer a clue. When after ligation of a coronary artery branch, infarction in the area distal to the branch is almost complete, then no enlargement of collaterals takes place no matter how long the wait. If, however, there is surviving myocardium in the area supplied by the artery, then collateral channels enlarge to an amazing degree, that is, roughly, proportional to the amount of surviving muscle and may, as I have said, amount to one or two hundred times the cross section of the pre-existing collateral channels. Teleologically, it might be considered that the demand for blood determines the ultimate degree of enlargement of the collateral channels. I think it would be best to say that there are probably metabolic factors in the partially ischemic zone of muscle that exert a profound influence upon the adjacent collateral arterial channels. I think there are great unexplored areas in metabolism of partially ischemic tissue, and Goldblatt's work on the kidney is a brilliant example of such an altered metabolism.

These little collateral arterial channels that man can now make, in addition to those that are already there, may seem of little or no functional value to the physiologist. When we find better ways of aiding them to grow, that will be of the greatest significance.

DR. EMILE HOLMAN, San Francisco.—May I express great skepticism that anyone will ever be able to convert a vein into an artery. Mont Reid made an attempt to reverse the circulation in the kidney but it resulted only in a fibrotic and functionless kidney.

DR. ARTHUR M. VINEBERG, Montreal.—These people have a very slim margin. The patients one would operate on are those who are no longer enjoying life; none of them would be able to take a long walk twenty years later. Many are totally incapacitated. In the surgical cardiac clinic at the Royal Victoria Hospital, we have had many cases with the coronary arteries practically occluded in which there was a very small stream of blood supplying the ventricle. Simple ligation of the great cardiac vein involves slight tipping of the ventricle, and the pushing of the auricle to one side. When this is done, we have seen the blood pressure drop rapidly to dangerously low levels.

I do not see how anastomosis can be made to the coronary sinus in its present position, without great harm to the patient. If we are going to operate on these people we have to do so quickly and by a simple procedure. We are not ready to go ahead with human patients as yet, but this operation is extremely simple. I would like to show one slide to show how the artery lies in position within the ventricular muscle.

COMPLETE FAILURE OF STERNAL FUSION WITH HERNIATION OF PERICARDIUM

Report of a Case Corrected Surgically in Infancy

Herbert C. Maier, M.D., New York, N. Y., and Frank Bortone, M.D., Jersey City, N. J.

EXTENSIVE malformations of the sternum which are of clinical significance in the neonatal period are very rare. Minor congenital anatomic variations are occasionally found. Complete failure of sternal fusion is usually associated with other serious anomalies, such as ectopia cordis, which may be incompatible with life. We are reporting a case of extensive maldevelopment of the sternum associated with protrusion of a part of the pericardium through the thoracic wall defect. Surgical correction of the abnormality was successfully accomplished. This case demonstrates the importance of surgical intervention shortly after birth if a normal thoracic cage is to be attained.

An extensive study of the development of the sternum from both the ontogenic and phylogenic standpoint has been made by Hanson,[2] and his conclusions have been most widely accepted. According to him the sternum first arises as two laterally situated sternal bands independent of the ribs (Fig. 1). These sternal bands are already present in the 6-week-old embryo. At the cephalad end a single median anterior rudiment which is intimately associated with the shoulder girdle also develops. Gradually there is approximation of the sternal bands with the ventrally growing tips of the ribs. Simultaneously the two sternal bands fuse with each other. The fusion of the sternal bands is usually complete by the ninth week of embryonic life. There is also concurrent union of the sternal bands with the anterior sternal rudiment. Subsequently transverse divisions of the cartilaginous sternum result in a number of sternebrae. These lines of division or sutures always occur opposite the ends of each pair of ribs. Ossification of these intercostal sternebrae occurs with the appearance of one or more centers for each segment. At birth the sternum is cartilaginous except for small areas of ossification.

CASE REPORT

A. L., a female infant, was delivered at the Margaret Hague Maternity Hospital on June 24, 1948.* The birth weight was 3,250 grams. The baby cried spontaneously, and respiration and circulation seemed normal. The physical findings were normal except for the thoracic abnormality. When the infant cried there was bulging of intrathoracic structures through a defect in the midline of the anterior aspect of the thoracic cage. On palpation complete lack of fusion of the two lateral components of the sternum was evident. There was but slight separation of the caudal ends, whereas the upper ends of the sternum were about 1.5 cm. apart. As a result there was an increase in the width of the upper portion of the thorax, the chest having a contour more like a cylinder than a truncated cone. The sternocleidomastoid muscles

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Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

*Referred to us through the courtesy of Dr. Samuel A. Cosgrove.

assumed an almost vertical position because the inner ends of the clavicles were displaced laterally to a considerable degree (Fig. 2). The ribs ends had apparently fused with the lateral sternal bands in a normal manner. Cardiac pulsation was visible and could be readily felt

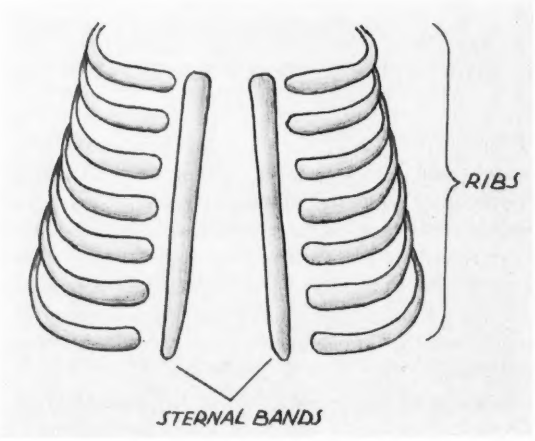

Fig. 1.—Diagrammatic presentation of sternal development in the 6-week-old embryo. There are two sternal bands which have not yet fused with the anterior ends of the ribs.

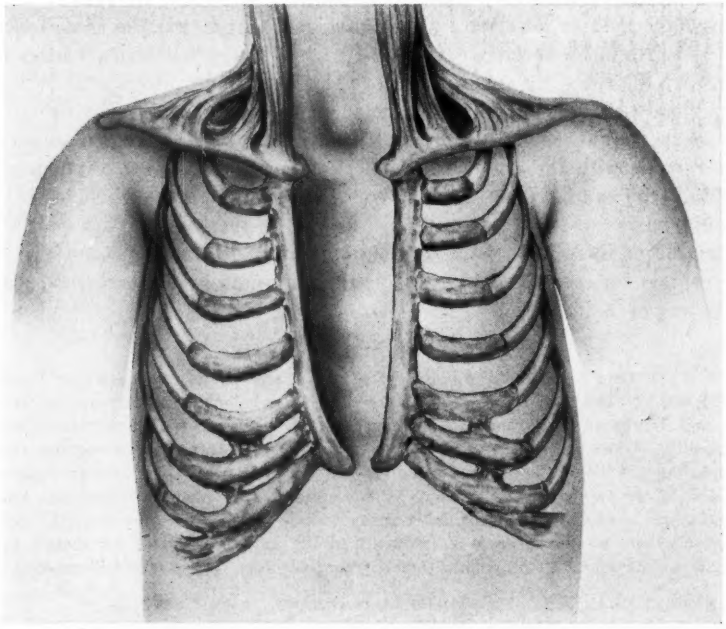

Fig. 2.—Type of sternal maldevelopment present in reported case. The sternal bands are completely separated. A wide gap is present between the inner ends of the clavicles.

just beneath the skin in the lower part of the sternal defect. There was alternating retraction and bulging of the soft tissues through the sternal gap with inspiration and expiration (Fig. 3). These movements were quite marked when the infant cried or strained. There was no cyanosis or other obvious evidence of circulatory disturbance associated with the paradoxical motion of the thoracic wall at the site of the defect. There was no clinical evidence of congenital heart disease.

The only other abnormality noted was a midline defect of the derma extending upward from the umbilicus for a distance of 2 cm. Its appearance resembled that of a healed linear scar. No umbilical hernia was evident.

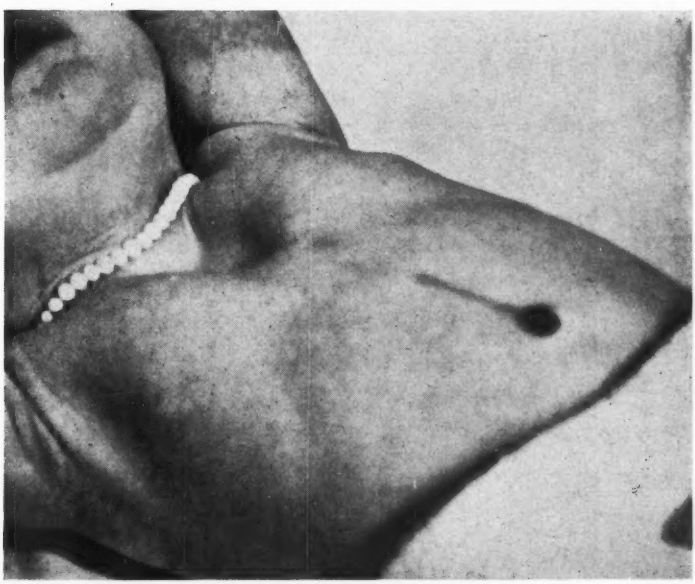

Fig. 3.—Photograph of infant at 2 weeks of age. Sternal defect indicated by retraction of chest wall in midline on deep inspiration.

Roentgenograms of the chest showed slight widening of the upper part of the thorax and of the mediastinal shadow, and elevation of the diaphragm (Fig. 4). The lung fields were clear. The clavicles appeared to be normal but retracted laterally. It was not possible to demonstrate the sternal defect itself since the sternum at birth is still almost entirely cartilaginous. The lateral view on deep inspiration revealed a marked retraction of the soft tissues of the anterior chest wall with posterior displacement of the trachea.

Operation was recommended when the infant was seen at the age of 2 weeks, but the procedure was not undertaken at once. We did not appreciate at the time the error that we were making in delaying operation. When first seen the sternal bands could almost be brought together in the midline by lateral compression of the thoracic cage. This maneuver did not cause any respiratory or other obvious embarrassment to the infant. Four weeks later, however, the thoracic cage was less flexible and the upper part of the defect wider, so that lateral compression no longer brought the edges of the sternal bands together with ease. The advisability of correcting the deformity without further delay was then very obvious.

The operation was performed on Aug. 11, 1948. Cyclopropane anesthesia was given, but the breathing was not satisfactory until the sternal bands could be brought together. Prior to that time the defect permitted so much paradoxical motion that the ventilation of the lungs

was inadequate. With a closed anesthesia system the infant's inspiratory effort tended to suck in the soft tissues at the site of the defect rather than satisfactorily aerate the lungs.

A midline incision was made from the lower cervical region to the epigastrium. After incision of the subcutaneous tissues a thin-walled sac containing clear fluid was encountered in the lower half of the wound. This sac was the pericardium which not only protruded through the sternal defect but also extended superficial to the thoracic cage and pectoral muscle on the left side. The heart itself did not protrude through the sternal defect except very

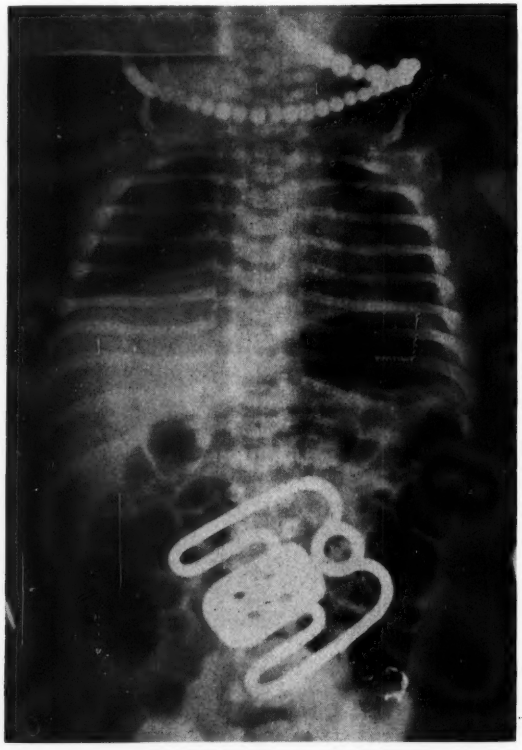

Fig. 4.—Preoperative roentgenogram; note wide mediastinal shadow and high position of diaphragm.

slightly when the infant strained. The pericardium might be described as having a large outpouching anteriorly with an associated increase in the amount of pericardial fluid (Fig. 5). The thymus bulged into the upper part of the wound but appeared otherwise normal. The lungs were not visualized, and neither pleural cavity was entered during the operation.

The herniated portion of pericardium was dissected from the subcutaneous fat, outer aspect of chest wall, and the edges of the sternal bands. The pericardium was opened during the dissection. Although there was a redundant portion of the pericardium, slight overlapping of the edges of the pericardial incision during its closure eliminated most of the excess. The thymus could be readily held within the thoracic cage. It was then possible by means of sutures to bring the sternal bands together without any other structures protruding between them. Since each sternal band was curved inward at its caudal end, the lower ends abutted on each other before the upper ends came together when traction was made with sutures.

Therefore, the cartilaginous part of the sternal bands on each side was divided transversely at the level of the fourth interspace so that the inward curve at the caudal end could be eliminated (Fig. 6). In so doing, care was taken to avoid complete division of the perichondrium laterally which would have interrupted the continuity of the sternum.

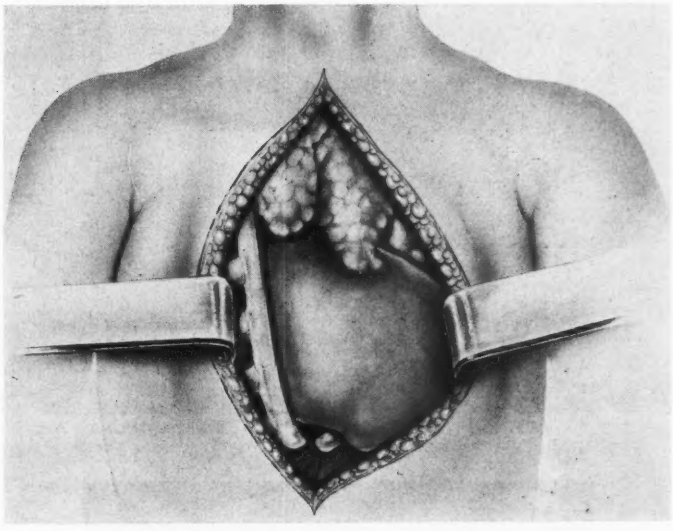

Fig. 5.—Operative findings. The herniated, redundant pericardium extends superficial to the left half of the sternum. Thymus is seen in the upper part of the incision.

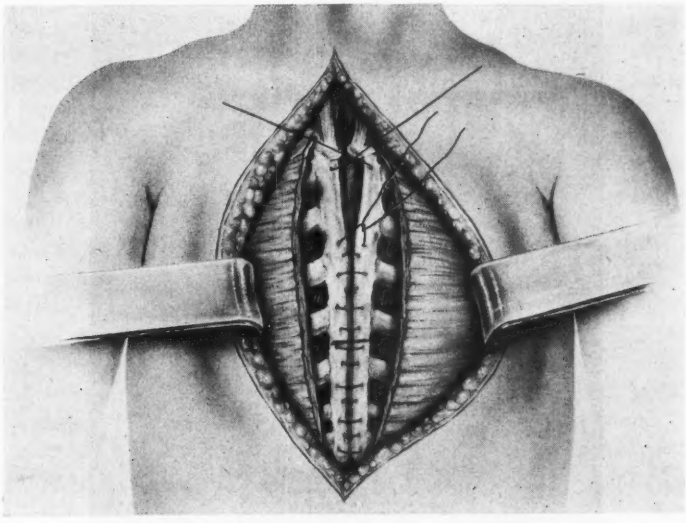

Fig. 6.—Operative procedure. The pericardium and thymus are now within the thorax, and the two sternal bands are being brought toegther with interrupted sutures. A partial transverse division of the sternal cartilage at the level of the fourth interspace has aided alignment.

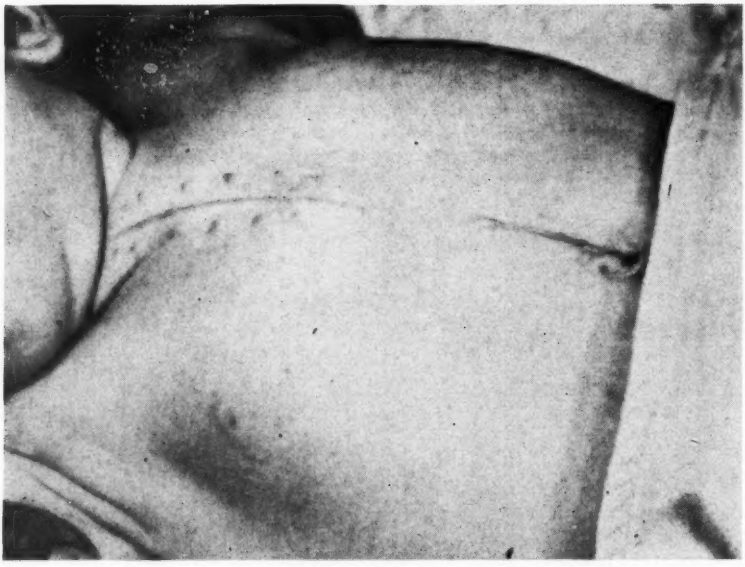

Fig. 7.—Photograph of infant two weeks postoperative. Deformity has been corrected.

Fig. 8.—Postoperative roentgenogram. Contour of thoracic cage is now normal.

The two sternal bands covered by perichondrium were brought together and held in the midline with a series of interrupted silk sutures. The first few sutures of 00 silk were tied with some tension which, however, was relieved by additional sutures. The stitches were inserted through the middle of the sternal band on each side. Thus, perichondrium was brought in apposition to perichondrium. A satisfactory closure was obtained. The pectoral fascia was brought together with very fine silk in the midline as an additional layer. The skin, which was slightly redundant, was closed with interrupted silk. The baby's condition was good throughout the operative procedure.

The postoperative course was uneventful. No respiratory or circulatory difficulties arose. The temperature was normal on the second postoperative day. The wound healed by primary union. No separation of the sternal components occurred at any time following the operation. Roentgenogram of the chest revealed no abnormal findings. The contour of the thoracic cage is normal (Figs. 7 and 8). The baby was developing well when seen at 8 months of age.

DISCUSSION

After observing the changes which occurred in the thoracic cage of this infant between the second and sixth week after birth, we feel certain in retrospect that the operative procedure should have been performed at an earlier date. At 2 weeks of age the lateral components of the sternum could be brought together rather easily by gentle manual compression of the shoulders toward each other anteriorly so as to bring the inner ends of the clavicles together. This was no longer possible when the operation was performed at 7 weeks of age. The shape of the defect had also changed by this time. At birth the width of the gap between the upper end of the lateral components of the sternum was only slightly greater than the width of the gap at the lower end. By the sixth week, however, the upper ends were more widely separated, whereas the lower ends were perhaps slightly closer together so as to produce a V-shaped defect. It seems that the weight of the shoulder girdle transmitted through the clavicles accounted for this change. The normal intrauterine position with the shoulders rotated forward across the chest probably eliminates much traction on the lateral parts of the nonfused sternum prior to delivery. After birth, however, the infant was lying on her back most of the time, and such a position would tend to increase the separation.

Prior to operation we were concerned over the effect upon the heart, great vessels, and air passages of bringing the sternal edges together. Subsequent developments showed that in this particular case the intrathoracic structures accommodated readily to the correction of the thoracic cage defect.

A thorough review of the literature on cleft sternum and ectopia cordis was made by Greig.[3] The extensive bibliography which that article contains lists the references on cleft sternum appearing before 1926. He analyzed 39 reported cases of cleft sternum in which clinical data were available in more or less detail. In 20 of these cases varying degrees of nonfusion of the component parts of the sternum were present without cardiac displacement. In an additional 19 cases cleft sternum was associated with ectopia cordis. There were 15 further cases of ectopia cordis but without cleft sternum; in 13 there was abdominal ectopia cordis, while in the remaining 2 cases cervical ectopia was present. A few instances of prolonged survival with an intra-abdominal heart are on record. No patient with cleft sternum associated with ectopia cordis has lived for more than a few weeks.

Since the sternum may be entirely absent without a true ectopia cordis, it is obvious that nonfusion of the sternum is not necessarily related to the displaced position of the heart. In cases of an antethoracic position of the heart, however, a defect of the sternum must also be present. In some of these there may be only a relatively small opening in the sternum through which the great vessels pass. There is frequently no skin and no parietal pericardium over the exposed heart. In our case there was herniation of a redundant pericardium through the sternal defect. This factor in itself would have prevented fusion of the lower part of the sternal bands.

Nonfusion of the sternal bands may erroneously be described as absence of the sternum. If the sternum is truly absent, the inner ends of the cartilages would not be joined to each other by a cartilaginous bar, since the latter is a part of the sternal anlage. Only post-mortem or operative examination can distinguish definitely between absence of sternum and nonfusion of sternal bands in infancy.

We have been unable to find a report in the literature of successful correction of complete failure of sternal fusion in infancy. There are only a relatively small number of cases of complete fissure of the sternum recorded. In most instances death occurred shortly after birth. A few patients survived for many years, but in most such cases there was very little separation of the two lateral components of the sternum, and usually little more than a fissure was present. Of the very few adults with some separation of the two halves of the sternum, the degree of cardiorespiratory impairment varied from slight to moderate.

Burton[1] reported two cases of congenital nonfusion of the greater portion of the sternum. Operative intervention was undertaken at the age of 12 weeks in one case, whereas the other child was 12 years old at the time of the operation. No attempt was made to bring the edges of the defect together, but a free graft of cartilage was inserted into the defect. A segment of several cartilages comprising the costal arch was utilized as the graft. The procedures were undertaken in order to give protection to the heart, which was covered only by skin and subcutaneous tissue in the lower part of the defect. Only a fair cosmetic result was obtained. Although the author calls the condition one of ectopia cordis, his patients seemed to have sternal defects primarily.

SUMMARY

Complete failure of sternal fusion with a defect extending the entire length of the anterior thorax is a very rare occurrence. Such a congenital maldevelopment of the sternum may be associated with ectopia cordis. Due to the lack of a rigid thoracic cage at the site of the sternal defect, considerable paradoxical motion may occur with resultant respiratory and cardiocirculatory difficulty. If only a partial cleft sternum or a narrow sternal fissure is present, symptoms may be slight or absent.

The embryological development of the sternum is briefly reviewed. A case of complete lack of fusion of the entire length of the sternum with herniation of the pericardium is reported. This is apparently the first case reported in

which such a congenital defect was successfully corrected by surgery in infancy. The importance of performing the operation very shortly after birth is well illustrated by our case. The relationship between cleft sternum and ectopia cordis is discussed briefly.

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DISCUSSION

DR. KARL KLASSEN, Columbus, Ohio.—On May 9, 1948, we performed a successful surgical repair of a cleft sternum with partial ectopia cordis on a 4-hour-old twin infant weighing 4½ pounds. We would like to present a short motion picture which illustrates several points brought out by Dr. Maier. (Motion picture.)

There is marked separation of the sternal plates with protrusion of the pericardium. The coronary vessel can be seen through the skin defect. X-ray films taken before operation show complete absence of ossification in the sternum, which was present in the normal twin. A T-shaped incision was used with resection of redundant and necrotic skin over the pericardium. Repair was made similarly to the method described by Dr. Maier. Approximation of the cartilaginous plates was accomplished without difficulty. At 2 weeks this infant appears to be normal.

The boy did well until the age of 8 months, when there again appeared separation of the sternal plates. Ossification in the sternum was still absent. Because of cardiac arrhythmia and apparent pain on movement, we performed a second operation on Jan. 24, 1949. The medial edges of the cartilaginous plates were exposed, the perichondrium and irregular cartilage removed and four tantalum pegs inserted into the substance of the sternal plates, following which silk sutures were used to approximate the two portions of the sternum. There was no change in the baby's pulse. On completion of the operation the infant appeared in good condition; however, when he was transferred to his bed, he died suddenly.

Autopsy showed a normal infant. There was no cardiac abnormality.

DR. MAIER.—Of course we do not know about the final development of the sternum in this child. When seen at the age of 8 months the external appearance was normal, and a firm structure without fissure was present. We do not, as yet, have roentgen evidence of ossification.

ANGIOCARDIOGRAPHY AND AORTOGRAPHY IN THE DIAGNOSIS OF CONGENITAL CARDIOVASCULAR LESIONS

THOMAS H. BURFORD, M.D., MERL J. CARSON, M.D. (BY INVITATION), AND WENDELL G. SCOTT, M.D. (BY INVITATION)
ST. LOUIS, MO.

THE rapidly expanding scope of corrective surgery in congenital cardiovascular disease has emphasized the necessity for precise diagnosis. The contributions of many, but Gross and Hubbard,[1] Blalock and Taussig,[2] and Crafoord and Nylin[3] in particular, have in a very short period revolutionized the attitude of the entire profession in regard to these lesions. Fortunately, these patients are no longer crudely classified on the basis of blueness or a poorly described murmur and forgotten. Today they are salvaged, many times at the remotest levels of practice, and referred for evaluation and treatment.

Since at present the surgery of these defects is limited to definite types of deformity, and since the margin of safety is probably less in this category, particularly if the operation does not achieve correction, than in any other group of patients, the concept of an exploratory operation, valid as it is in other procedures, is rarely defensible.

As those experienced in the management of these cases known, a great many, probably the majority, can be confidently diagnosed by simple examination and fluoroscopy. There is, however, a very significant borderline group in which more exhaustive studies are necessary. Physiologic studies, such as cardiac catheterization, measurements of pulmonary capillary flow, oxygen consumption studies before and after exercise, and oximetry are of the utmost value. These studies, particularly cardiac catheterization as applied by Cournand and Ranges[4] and Bing and associates,[5] have been of the greatest importance in elucidating and even quantitating the hemodynamics of these anomalies. However, these procedures are intricate in their application and require elaborate laboratory facilities for their use as well as a full-time, highly trained staff. More important, the technique of venous cardiac catheterization is not consistently applicable, at least in our hands, in the study of infants and very young children. We are aware of Cournand's experience in this regard and will be interested in his remarks on this point.

Serial contrast visualization of the cardiac chambers and great vessels (intravenous angiocardiography) was proposed in infants by Castellanos, Garcia, and Pereiras,[6] and in adults by Robb and Steinberg.[7] Sussman and associates,[8] in particular, have had a large experience with the procedure. Despite the enthusiasm of these workers, the technique has not, until very recently, had as wide an application as we feel it merits. The direct information as to the course of intracardiac blood flow and relative size and location of the cardiac

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Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

chambers and great vessels which can be obtained by angiocardiography has been of the greatest value to us in establishing the exact diagnosis in borderline cases of congenital heart disease. It has been of particular value in differentiating the various types in which cyanosis is a prominent symptom. The method has the very great advantage of being applicable to patients of all ages; diagnostic serial radiograms are obtained as easily in small infants as in children and adults.

Successful angiocardiography requires two conditions. First of all, a sufficient volume concentration of dye (70 per cent Diodrast or 75 per cent Neoiopax) must be moved rapidly into the zone to be visualized. This requires the very rapid intravenous injection of 40 to 50 c.c. of the dye in adults, and proportionately lesser amounts in children and infants. The needle size should be only slightly smaller than the vein used. Second, it is necessary to have equipment capable of producing a rapid sequence of radiographic exposures accurately synchronized with the presence of the dye in the heart and great vessels. Such newer automatized radiographic techniques as the tautograph,[9] developed by Wendell Scott in our Department of Radiology, make this possible. An improved model which we are now using is called the rapidograph[10] and utilizes a modification of the aerial camera of the Fairchild Camera and Instrument Company. With this camera, exposures are taken every two-thirds or one-half second, as desired.

When these two conditions are met, consistently good angiocardiograms are obtained by the intravenous method showing the heart and the pulmonary arteries. However, with dispersion of the dye through the pulmonary circuit and return to the left heart, there is frequently very poor delineation of the aorta and its major branches. For this reason it has been felt that injection of radiopaque material directly into the aorta would yield more clearly defined pictures of the aorta and its immediate branches. Dos Santos and associates,[11] and later Wagner,[12] had advocated direct puncture of the aorta through the paravertebral region. Fariñas,[13] on the other hand, had catheterized the femoral artery in Scarpa's triangle and passed a catheter to the desired level. Crafoord[14] and others have catheterized the brachial artery and successfully visualized the aorta by retrograde injection of dye. Finding the intra-arterial catheterization technique difficult, we decided to use the left carotid, and found after employing this method that Freeman[15] quite independently was utilizing a similar technique. After a moderately extensive trial, we are convinced that the retrograde arterial technique is a valuable method of diagnosis and evaluation in those cases where the lesion is of the aorta or its immediate branches. We are further convinced that introducing the dye into the carotid is simpler and superior to other techniques.

By this technique the common carotid artery, usually the left, is surgically exposed and provision made by loose ligatures to introduce a large cannula in a bloodless fashion. When the cannula is to be introduced, the distal tie is snugged down so that the dye cannot go into the cerebral circulation. The proximal tie is then brought down tight enough to shut off carotid blood flow

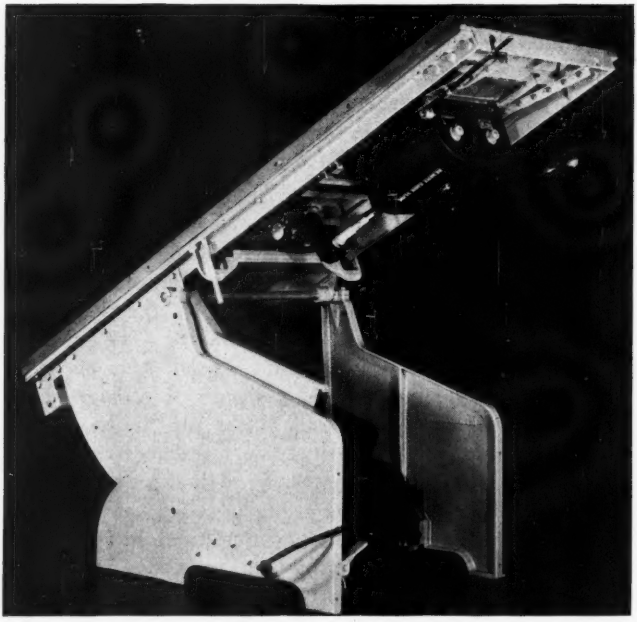


Fig. 1.—View of rapidograph table showing position of modified aerial camera. In this photograph, rapidographic table is tilted. Angiograms and aortograms are done with table flat.

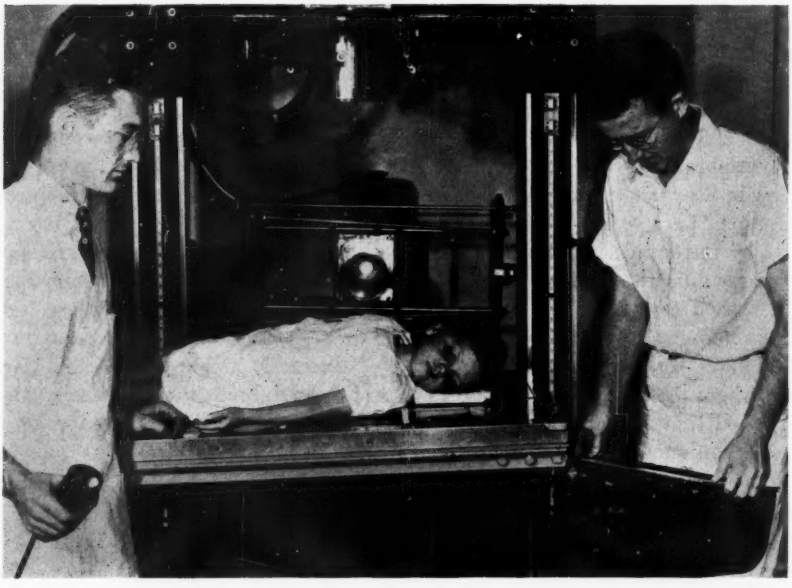
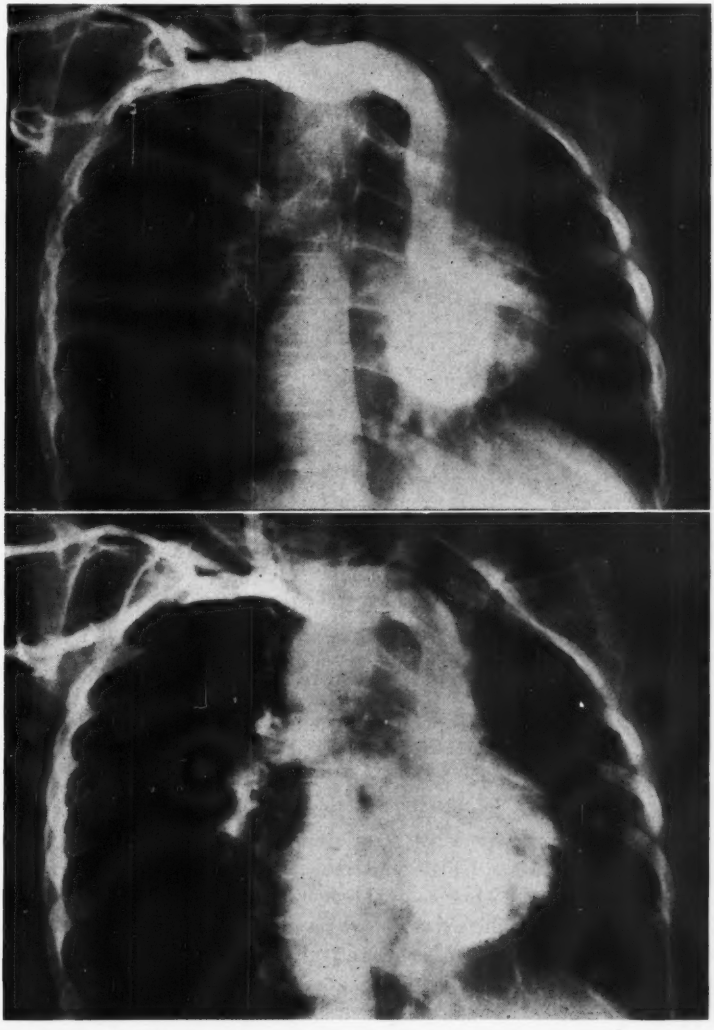


Fig. 2.—Showing complete assembly of rapidograph with patient in position for intravenous injection of Diodrast. Radiologist at right is preparing to put the loaded camera into position.

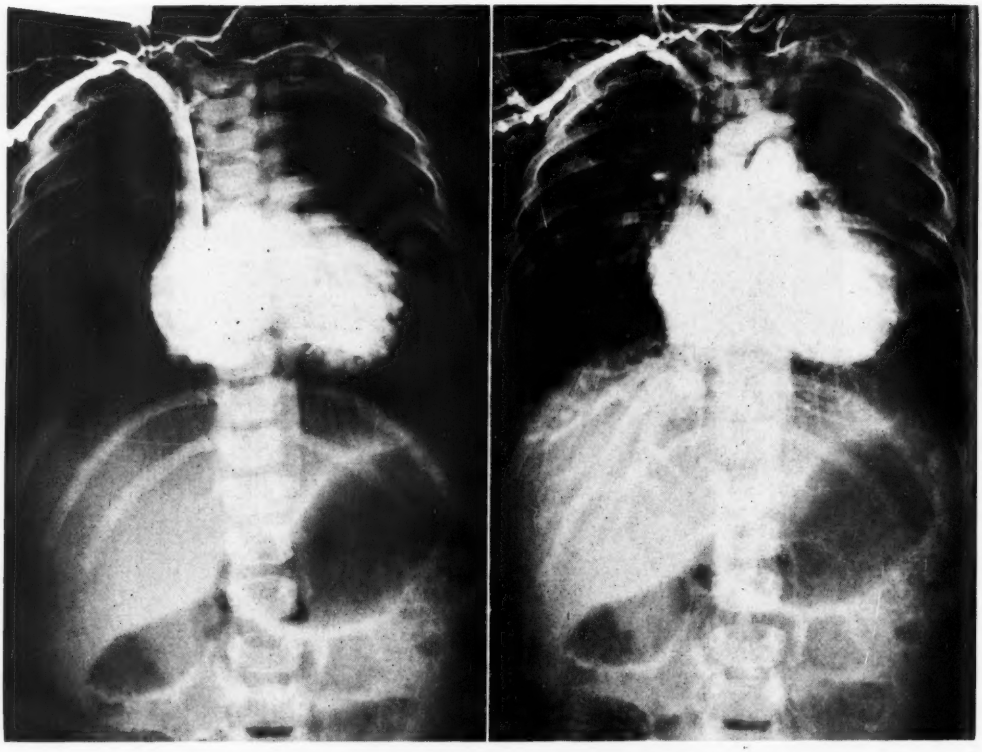
and the cannula introduced toward the heart through a small incision in the artery between the ties. When the cannula is introduced, a third ligature is tied over it to prevent leakage, and the proximal tie is released. The dye is then pushed in very rapidly by means of a large syringe. When the exposures have been completed, the proximal ligature is again tied and the needle removed. The opening in the artery is carefully repaired with 00000 Deknatel and the neck wound closed with silk.



Figs. 3 and 4.—Two films from the angiocardiographic series of a case of tetralogy of Fallot showing the typical findings. These reproductions are reversed so that the right side is to the left and vice versa. There is clearly demonstrated the filling of the left ventricle across a high septal defect with simultaneous filling of aorta and pulmonary artery. The pulmonary artery is significantly reduced in size.

By this method we have gained decisive information in cases of coarctation of the aorta, patent ductus arteriosus, aortic arch anomalies, and aneurysm.

In cases of coarctation, not only is the diagnosis made, which admittedly is not often too difficult, but the site and type are delineated. This latter information is of the greatest value in preoperative planning.



Figs. 5 and 6.—Angiocardiograms of a case of nonfunctioning right ventricle showing failure of the right ventricle to fill. The flow of the dye is from the superior vena cava to the right auricle, then to the left auricle and into the large left ventricle which gives rise to both the aorta and the pulmonary artery. Note how the latter structures fill simultaneously. The pulmonary arteries are smaller than normal. There is marked reflux into the hepatic veins.

In the infrequent but important borderline case of patent ductus arteriosus, retrograde visualization is decisive. The simultaneous opacification of sorta and pulmonary artery or the actual demonstration of the ductus are irrefutable evidence of ductal patency. Absence of these evidences of shunt can be confidently interpreted as excluding the existence of a patent ductus.

Aortic arch anomalies can be beautifully outlined even to the extent of showing the two limbs of an "aortic" ring.

Aneurysms can be visualized by actual filling or partial filling. In those that are filled with clot, there is enough suggestive evidence in the aortogram to make the diagnosis.

Since developing the tautograph and the rapidograph, we have done eighty-six visualizations. Of these, sixty-three were intravenous angiocardiograms and twenty-three were retrograde aortograms. So far as we have been able to discern from operations and post-mortems on this group, there has been but one wrong diagnosis.

Fig. 7.—Film showing characteristic typical angiocardiographic features of an Eisenmenger's complex. Here again there is overriding with simultaneous filling of pulmonary artery and aorta and evidence of a high interventricular septal defect. The pulmonary artery is, however, larger than normal.

To date we have had no deaths or untoward event escribable to the dye or the method. Two deaths have occurred following angiocardiography, but in both instances the patients were very young infants and were extremely poor risks. Death was not unexpected. Needless to say, all patients are tested for possible sensitivity to the dye beforehand.

In the retrograde group there have been no complications attributable to the use of the carotid artery.

In conclusion, we would stress that angiocardiography is not to be recommended in every case of congenital cardiovascular heart disease. Despite our experience and the larger experience of others, it is not a method without some potential hazard. Moreover, as we said in the beginning, the diagnosis in most

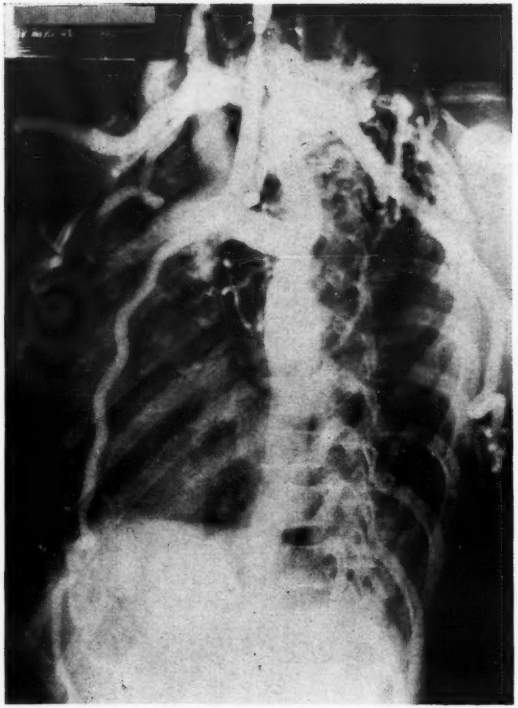


Fig. 8.

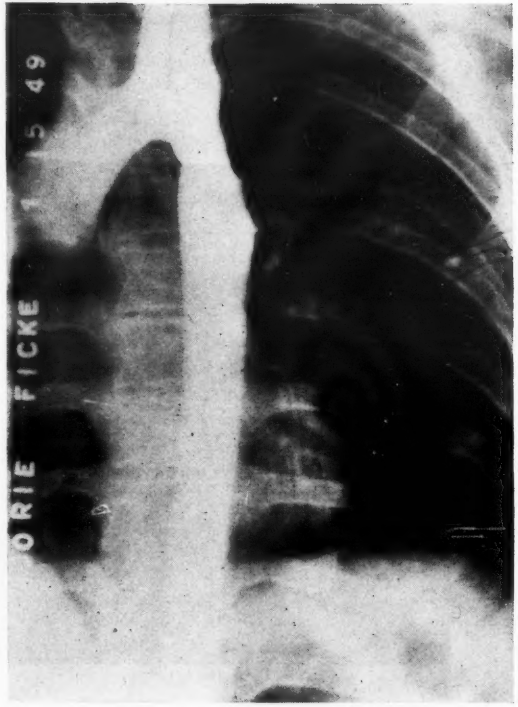


Fig. 9.

Fig. 8.—Aortogram done by the retrograde technique showing visualization of a typical case of coarctation of the aorta of the adult type. The lesion is accurately localized and valuable information is obtained concerning the length of the constricted segment. Note the enormous collateral channels.

Fig. 9.—Retrograde aortogram made one year after resection and anastomosis of aorta for coarctation of the aorta. Note the normal-sized lumen of the aorta and the regression of collateral channels.

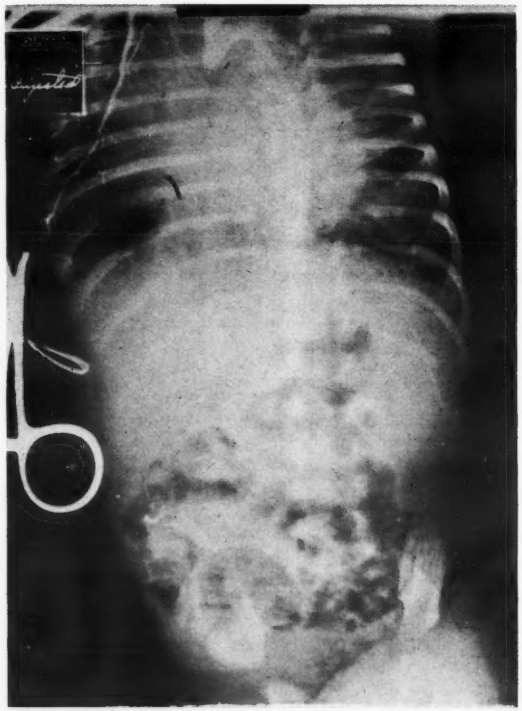


Fig. 10.

Fig. 11.

Fig. 10.—Retrograde aortogram of coarctation of the aorta of the infantile type. Note the long atretic segment.

Fig. 11.—Retrograde aortogram of aortic arch anomaly. Careful examination reveals the presence of aortic ring.

instances can be confidently arrived at by simple examination and fluoroscopy. Nor would we imply that visualization should supplant physiologic studies when these are indicated and obtainable.

In the all-important borderline group, however, we fieel that angiocardiography and retrograde aortography when properly done offer a reasonably simple, a very safe, and a very acurate method of diagnosis that is applicable to all age groups.

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(See discussion on page 891.)

THE ROLE OF ANGIOCARDIOGRAPHY AND VENOGRAPHY IN MEDIASTINAL AND PARAMEDIASTINAL LESIONS

OSLER A. ABBOTT, M.D.,* WILLIAM A. HOPKINS, M.D.* (BY INVITATION), AND TED F. LEIGH, M.D.† (BY INVITATION), ATLANTA, GA.

SINCE the original publication of Robb and Steinberg[1] in 1938, relative to visualization of the chambers of the heart, the pulmonary circulation, and the great blood vessels in man, the procedure of angiocardiography has become of increasingly widespread use and application as a valuable diagnostic procedure. The fundamental interest in this presentation is to evaluate the results of our experience in using this procedure as a diagnostic aid in the differential diagnosis of tumors in the mediastinum and paramediastinal areas. Visualization of both innominate veins and the superior vena cava, by means of contrast media, has also been used as an aid in the differential diagnosis of these lesions as well as to determine the status of these veins in patients having primary carcinoma of the lung.

It is not our purpose to state any final conclusions in regard to the value of this procedure in the differential diagnosis, but rather to describe the results of our experience in a fairly large number of cases and to evaluate the lessons derived therefrom.

The use of angiocardiography in the differential diagnosis between tumors of nonvascular origin and vascular aneurysms has been a common practice in many clinics during the past few years. Attention was drawn to this method of approach by Sussman[2] in November, 1947. The value of this procedure in the differential diagnosis between aneurysm and other mediastinal tumors is quite apparent. This procedure also has demonstrated a considerable value in the differential diagnosis of intracardiac lesions and also in certain pericardial lesions.[3, 10] It is our opinion that the procedure may also be of considerable value in both nonvascular lesions of the mediastinum and certain paramediastinal diseases, in particular pulmonary malignancy.

In reviewing the results obtained in the series of patients which will be presented, we feel that there are five primary questions which need investigation. From the data obtained to date, a satisfactory answer to each of these questions is not yet obtainable. We feel that the problems, in order of their importance, appear to be as follows: (1) Angiocardiography as a method of differential diagnosis between vascular and avascular mediastinal and paramediastinal lesions. (2) How much diagnostic information can be obtained from this method in relation to nonvascular tumors in these areas? (3) What is the relative place of angiocardiography in the study of mediastinal and paramediastinal masses? (4) What are the special technical problems associated with

Read at the Twenty-ninth Annual Meeting of The American Association for Thoracic Surgery, New Orleans, La., March 29-31, 1949.

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venography and angiocardiography in the different types of mediastinal lesions encountered? (5) Of what value can angiocardiography be in the study of certain primary pulmonary lesions, in particular pulmonary neoplasms?

1. *The Role of Angiocardiography in the Differential Diagnosis Between Vascular and Nonvascular Mediastinal and Paramediastinal Lesions.*—The problem of the differentiation between intrinsic and transmitted pulsation of masses visualized on fluoroscopy is frequently encountered. The initial enthusiasm for the roentgenkymographic method of differentiation has proved a considerable disappointment. Certainly, angiocardiography has proved the best solution of this problem to date. Unfortunately, lesions of primary vascular origin are prone to contain large areas of blood clot which may forbid adequate definition by this method. However, the channels through this lesion will frequently show a sufficient distortion to be of diagnostic importance. Of equal value in the extravascular tumor can be the visualization of an area of extrinsic compression of a major vessel by the tumor in question. These findings are exemplified in the following patients.

CASE 1.—H. S., a 17-year-old college student, was found to have a superior mediastinal lesion in the right paratracheal area in the course of an x-ray screening project. Fluoroscopic examination showed evidence of pulsation in this mass, which was thought to be transmitted and appeared to be paradoxical in relationship to the pulsations of the ascending aorta. A marked change in the conformity of the mass is noted in comparing inspiration and expiration x-ray films of the chest. The lateral x-ray projection failed to give detailed definition of the posterior portions of this mass. A definitive diagnosis was only made upon obtaining satisfactory visualization of the innominate veins and superior vena cava by means of 70 per cent Diodrast as part of a complete angiocardiographic study. This revealed the presence of a large fusiform aneurysm of the right innominate vein and the superior vena cava. Irregularities on the surface of this lesion suggested the presence of daughter aneurysms. This diagnosis was confirmed at the time of operation, at which time the lesion was treated by the application of Polythene cellophane.

The angiocardiographic study was repeated six months following operation and showed definite evidence of decrease in the size of the lumen and thickening of the wall of the aneurysm. This patient has been reported in detail elsewhere.[4] (See Fig. 1.)

CASE 2.—K. L., a 24-year-old white man, had been treated on two previous occasions with deep x-ray therapy on the suppositional diagnosis of malignant lymphoma. On physical examination the patient demonstrated a very loud, to-and-fro, coarse murmur in the second interspace just to the left of the sternum. Fluoroscopic examination revealed the presence of a nodular mass in the region of the aortic knob and extending upward into the superior mediastinum, but there was no apparent evidence of pulsation.

Roentgenograms taken in the lateral projection showed what we felt to be a very characteristic diagnostic finding, namely, the presence of a shadow assuming the appearance of a small embryo. This pseudoembryo shadow seen in the lateral projection is felt, in this clinic, to be very strongly suggestive of a congenital aneurysm. The location of this shadow in the posterior mediastinal area suggested that the aneurysm arose from the aorta in contradistinction to the anterior position of such shadows presented by congenital aneurysms of the left pulmonay artery. This impression was confirmed upon detailed angiocardiographic examination, revealing the presence of a large multilocular aneurysm of the descending limb of the aortic arch. This finding was confirmed at operation. The lesion was also treated by the application of Polythene cellophane. (See Fig. 2.)

CASE 3.—T. L., a 32-year-old white man, presented a very similar history to patient K. L.; namely, he had undergone two courses of deep x-ray therapy on the presumptive

diagnosis of mediastinal lymphoma. The lateral x-ray projection showed the presence of a large pseudoembryo shadow in the anterior mediastinal area, and angiocardiographic study confirmed the impression of a massive aneurysm of the pulmonary artery. This lesion was also confirmed at operation and treated by the application of Polythene cellophane. (See Fig. 3.)

CASE 4.—A. L., a 54-year-old white coal miner, had suffered from severe precordial and bilateral arm pain for sixteen months. The presumptive diagnosis had been recurrent

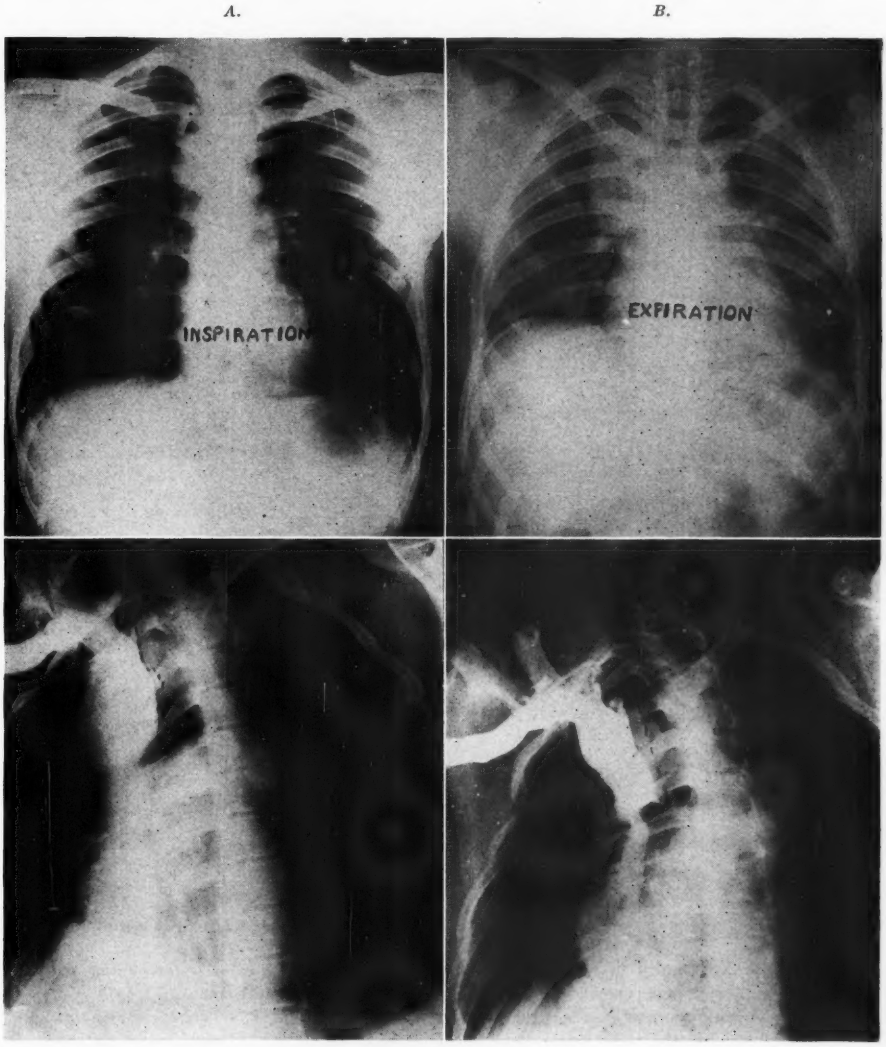
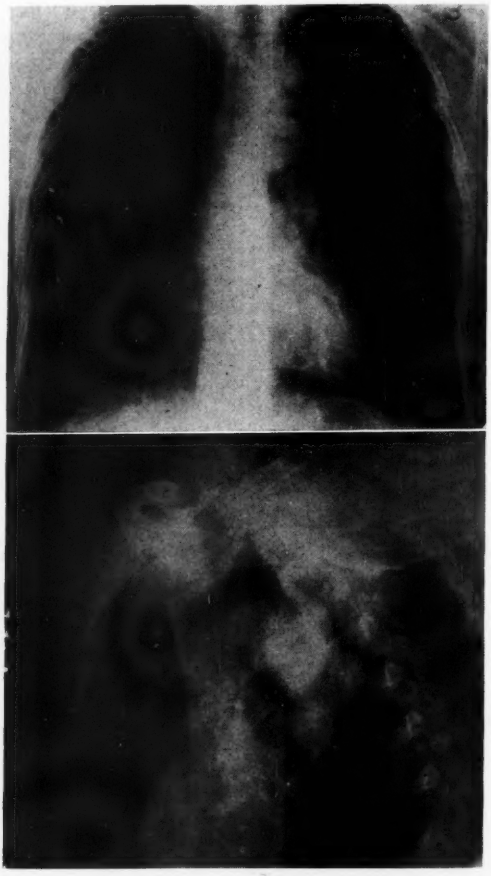


Fig. 1, *A* to *D* (Mr. H. S.).—Right paratracheal mass. Angiocardiogram showed this to be an aneurysm of the vena cava. *D* shows angiocardiogram six months after operation.

coronary occlusion and angina pectoris. He had undergone recent study at another clinic, which revealed the presence of a mass with apparent pulsations in the region of the ascending aorta. He was referred to this clinic with a tentative diagnosis of aneurysm of that structure.

Fluoroscopic examination certainly suggested intrinsic pulsation. There were no abnormalities on examination of repeated electrocardiographic studies. The extravascular character of the lesion could be defined only by means of the use of the angiocardiogram, which

A.



B.

Fig. 2, *A* and *B* (Mr. K. L.).—Showing large multilocular aneurysm of aorta.

revealed no abnormality in the superior vena cava, cardiac chambers, or ascending aorta. There was no apparent compression of the ascending aorta by this lesion.

Exploratory thoracotomy revealed that the primary shadow had been produced by an adenocarcinoma of the right lower pole of the thymus gland. The presence of metastatic lymph nodes invading the wall of the ascending aorta was considered to be the explanation for the patient's persistent pain. This pain was relieved by an extensive upper dorsal sympathectomy. (See Fig. 4.)

CASE 5.—I. A. R., a 48-year-old white man, had a cough of two weeks' duration, and x-ray examination revealed the presence of a very large mass occupying the left superior paramediastinal region.

Physical examination revealed definite bulging of the second and third left anterior interspaces with visible and palpable pulsations. The clinical impression at that time was massive aneurysm of the aorta. Fluoroscopic examination strongly suggested the presence

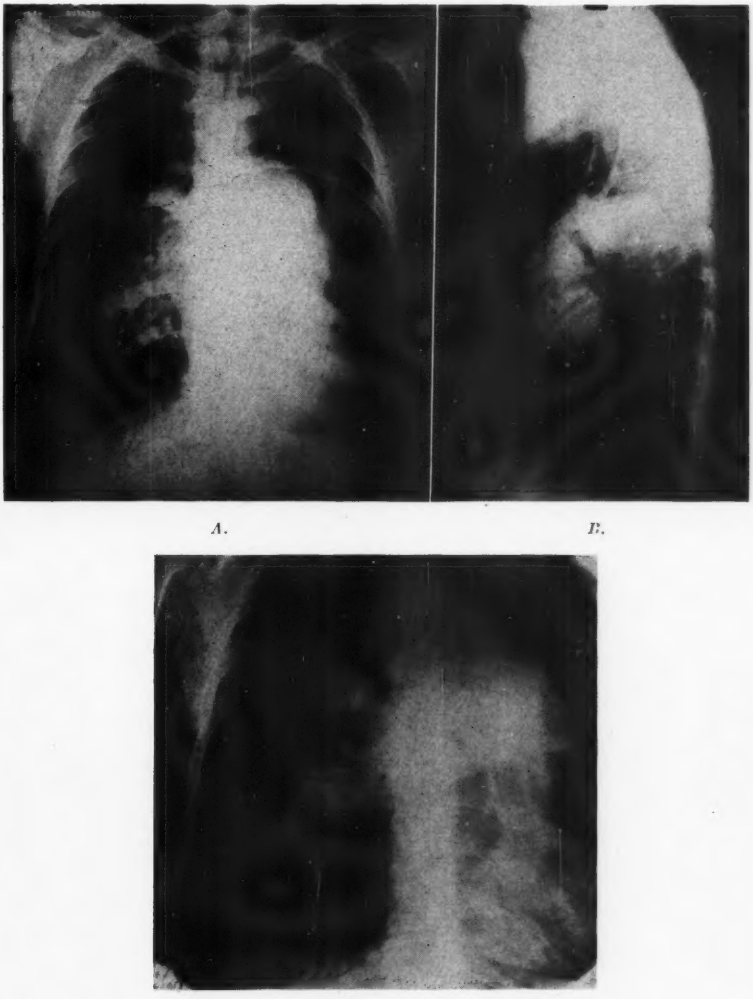


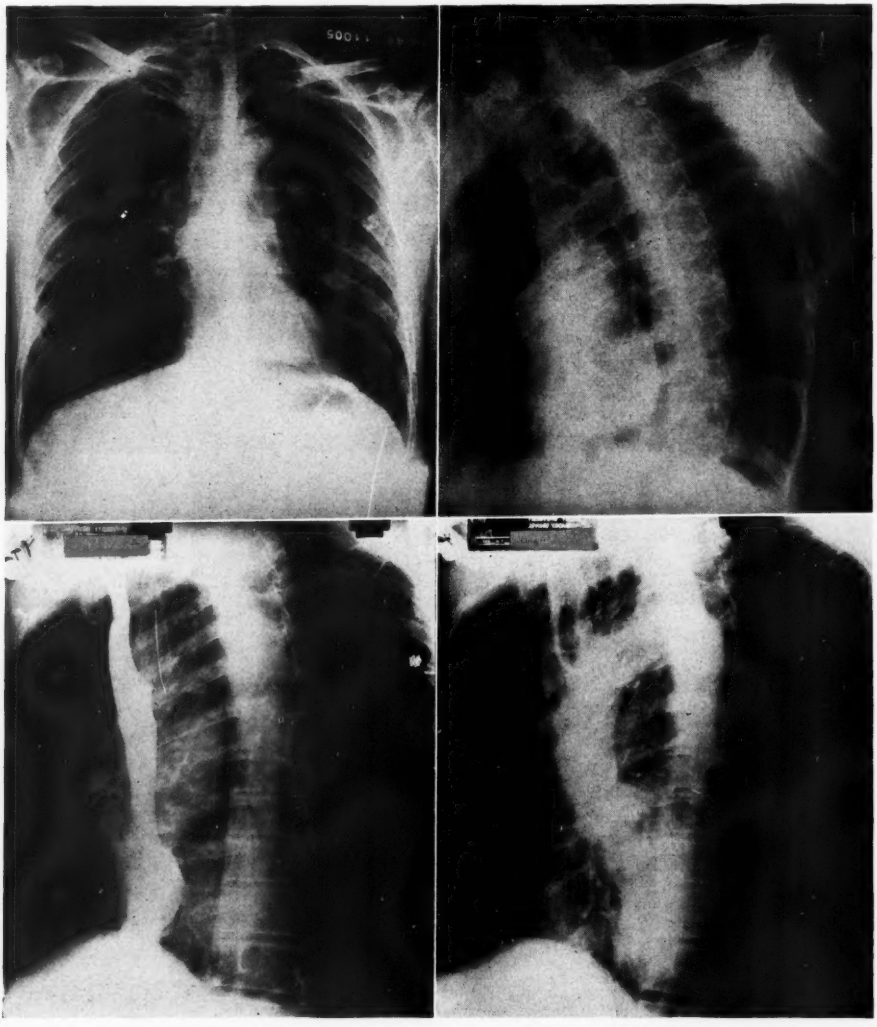
Fig. 3, *A* to *C* (Mr. T. L.).—Large aneurysm of the pulmonary artery. Note, on lateral x-ray, pseudoembryo formation. Angiocardiogram confirms diagnosis.

of intrinsic pulsation of the mass. A lateral x-ray projection showed the presence of an apparent cleft in the inferior aspect of the mass and an area of posterior nodular projection thought to suggest daughter aneurysm. Angiocardiographic studies revealed a very definite distortion of the vascular pattern in the apical portion of the left upper lobe; when the

aorta was visualized, there was no evidence of aneurysm, but rather, a definite area of extrinsic compression was demonstrated. The working diagnosis, therefore, was changed from aortic aneurysm to primary pulmonary carcinoma. The patient was treated by left total pneumonectomy which revealed a squamous carcinoma of the left upper lobe. (See Fig. 5.)

CASE 6.—G. T., a 56-year-old white man, had a history of an episode of extremely severe pain extending from the left anterior part of the chest through to the back, nine

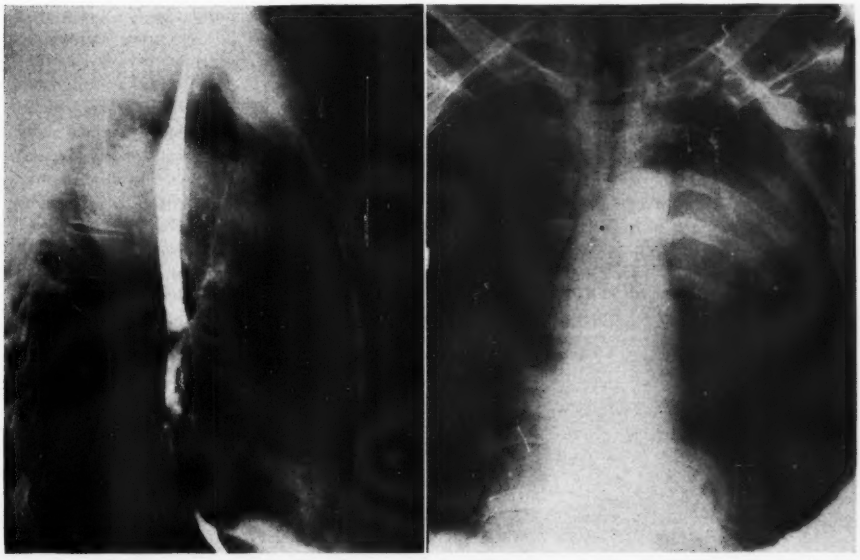
A. *B.*



C. *D.*

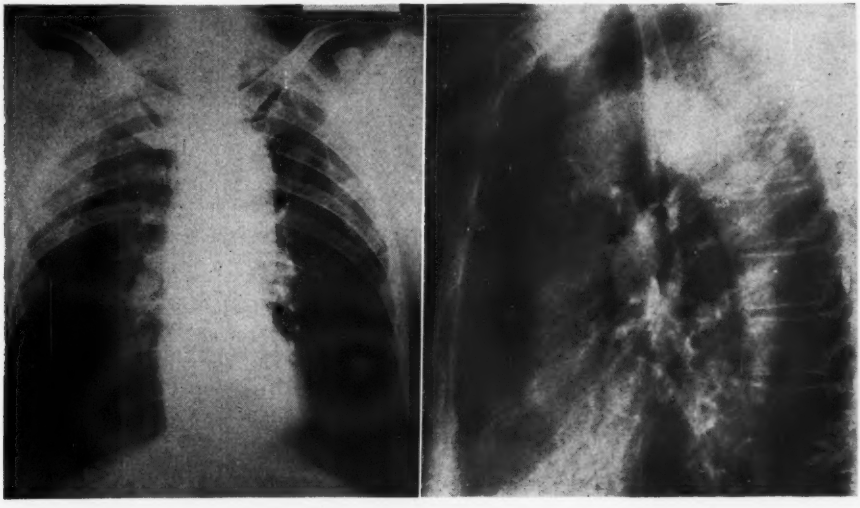
Fig. 4, *A* to *D* (Mr. A. L.).—Malignant thymoma. Angiocardiogram showing lesion to be entirely extravascular.

weeks prior to consultation. No unusual physical findings were present, and x-ray examination revealed the presence of a large mass occupying almost the entire extent of the posterior mediastinum. This lesion was suspected of being a dissecting aneurysm, and confirmation



A. *B.*

Fig. 5, *A* and *B* (Mr. I. A. R.).—Angiocardiogram *A* shows evidence of lateral compression of aortic knob. Diagnosis was carcinoma of the left upper lobe.



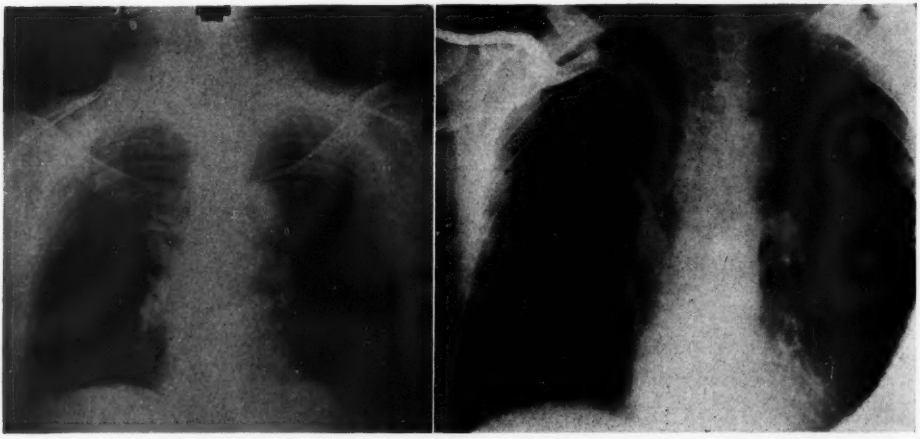
A. *B.*

Fig. 6, *A* and *B* (Mr. G. T.).—Angiocardiogram shows double channel effect on lateral view. This patient had dissecting aneurysm of aorta.

was obtained by angiocardiographic studies. This patient has been presented in detail elsewhere to emphasize the presence of a double channel on angiocardiographic studies of these lesions.[5] (See Fig. 6.)

CASE 7.—E. W., a 56-year-old white man, was seen with the presenting complaints of cough, fatigue, and vague discomfort of the upper part of the chest. X-ray and fluoroscopic studies revealed the presence of a rather poorly defined mass in the right paramediastinal area. The venous pressure studies were within normal limits. The differential diagnoses suggested involved partial atelectasis of the right upper lobe secondary to bronchiogenic carcinoma, mediastinal lymphoma, or tumor of vascular origin. Visualization of the right innominate vein, and superior vena cava by means of 70 per cent Diodrast readily demonstrated that the mass was medial to these structures, and there was no visible abnormality of these veins. The irregular contour of the lesion, plus its position medial to the major venous structures, led to a presumptive diagnosis of lymphosarcoma which was subsequently proved true.

It is felt that the value of this procedure in the differential diagnosis of lesions of this type needs no further discussion and is readily acceptable. (See Fig. 7.)



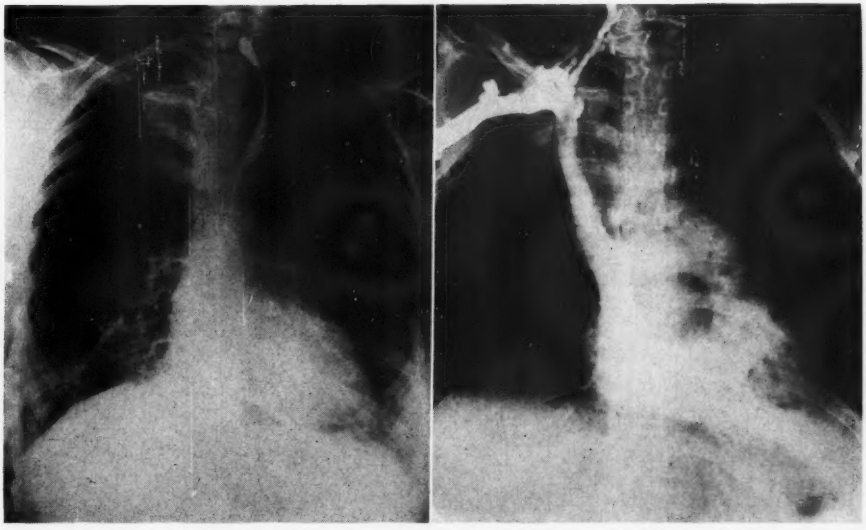
A. *B.*

Fig. 7, *A* and *B* (Mr. E. W.).—Showing marked lateral deviation of right innominate vein and vena cava. Diagnosis was mediastinal lymphosarcoma.

2. *How Can Valuable Diagnostic Information Be Obtained From Angiocardiography in Nonvascular Mediastinal and Paramediastinal Tumors?*—This is probably the most difficult question to be answered and the one to which the least satisfactory conclusions can be made at this time. It is felt that venography and angiocardiography can help in the localization of the borders of nonvascular mediastinal tumors. It is possible that on some occasions it may suggest the point of origin of the pedicle of these lesions. The frequency with which such information can be obtained can be determined only after obtaining considerably more information than is at present at hand. The basic information consists in obtaining evidence of vascular distortion of a specific pattern in relation

to certain primary lesions and, second, in the definition of vascular distortion due to invasive processes into the vascular structures either by the primary lesion or by metastatic neoplasm particularly in mediastinal lymph nodes. It is felt that there is a specific place for visualization of the superior vena cava when there are malignant lesions in the pulmonary system. The displacement of certain vascular structures is also felt to be suggestive help in determining the point of origin of a mass. Such information can also be of considerable value to the operating surgeon.

CASE 1.—R. W., reported upon in the foregoing section, was felt to be an illustration of the distortion of the right innominate vein and superior vena cava by involvements of the lymph nodes in the right paratracheal region by a malignant lymphoma. The lateral displacement of these structures limits the probable tissue of origin of this tumor to those structures which are normally contained between these vessels and the right tracheal wall. (See Fig. 7.)



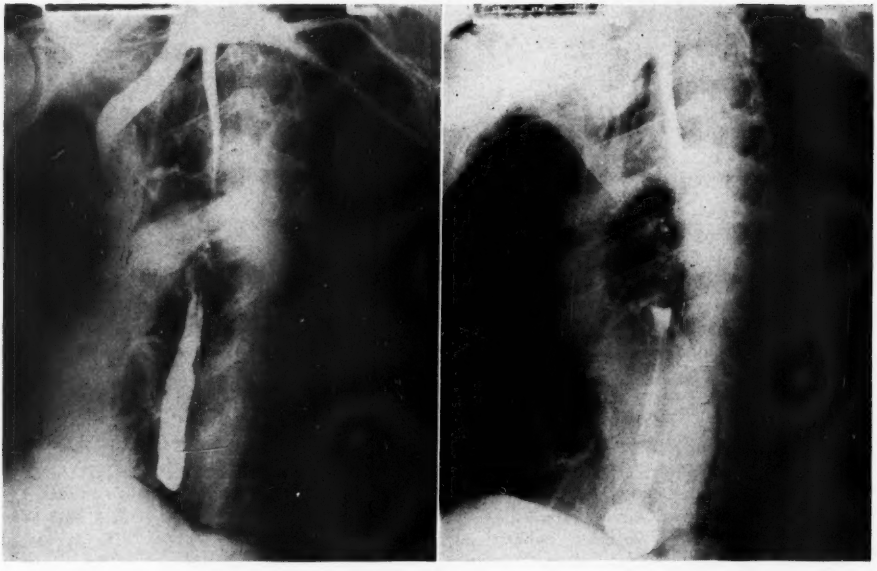
A. *B.*

Fig. 8, *A* and *B* (Mrs. R. T.).—Showing evidence of a lateral displacement of right innominate vein. Contrast dye injected through left side would probably have been of considerable help in localizing this lesion, as would a lateral venogram taken with dye in right innominate to outline compression of cava.

CASE 2.—R. T., a 49-year-old white woman, was admitted to the hospital with symptoms of tracheal obstruction. The patient was quite obese, and satisfactory palpation of the thyroid area could not be obtained, but it was felt that the thyroid gland was probably not abnormal. X-ray studies revealed the presence of a large rounded mass with a smooth contour occupying the superior mediastinum and deviating the trachea to the left. Angiocardiographic studies were carried out despite the presumptive diagnosis of substernal thyroid adenoma in order to evaluate this procedure in association with this lesion. The study did show a definite lateral deviation of the innominate vein on the right by the mass, but essentially no visible distortion of the superior vena cava on films taken in the antero-

posterior projection. Although the innominate vein was displaced laterally, there was no irregularity in its wall, and its smooth contour further suggested the possibility of a benign lesion. (See Fig. 8.)

A.

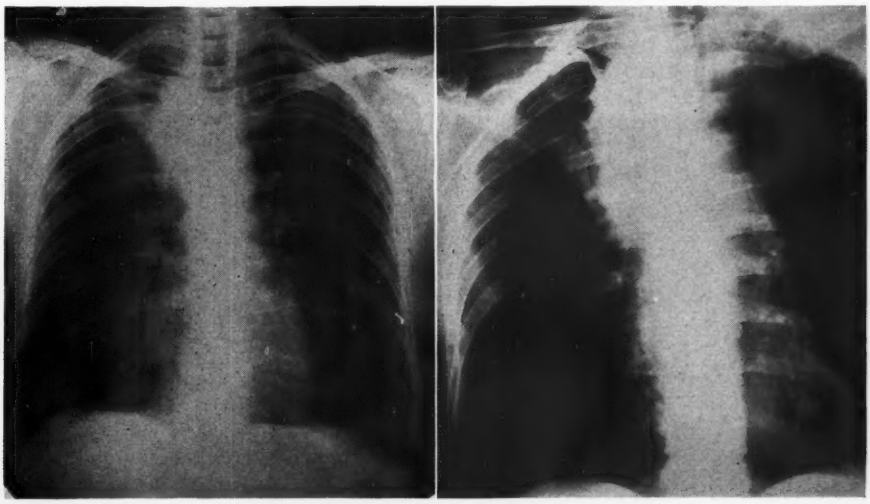


B. *C.*

Fig. 9, *A* to *C* (Mr. S. A. K.).—Note that the tumor compressing the esophagus is not related to any vascular lesion. Note the effect of associated filling of the esophagus with contrast media during angiocardiography.

CASE 3.—S. A. K., a 25-year-old white man, was admitted to the hospital because of symptoms associated with a duodenal ulcer. During the course of barium studies of the gastrointestinal tract, a major area of compression of the mid-portion of the esophagus was revealed. Following this discovery the patient related that he had had some dysphagia for several months. This area of compression was in the region of the major pulmonary vessels. Esophagoscopic examination revealed only evidence of extrinsic obstruction. Angiocardiographic study was carried out with the presence of barium in the esophagus. This study revealed no abnormality of the vascular bed, in particular, no enlargement of the left pulmonary artery. The diagnosis of nonvascular tumor of the esophageal wall was made, presumptively a leiomyoma. This was confirmed at operation. (See Fig. 9.)

CASE 4.—A. O. L., a 61-year-old white man, was seen with the complaints of fatigue, cough, and a vague discomfort of the upper part of the chest. X-ray examination revealed the presence of a right superior paramediastinal mass. Bronchoscopic examinations were noncontributory. Special studies did not reveal evidence of increased venous pressure. Diodrast visualization of the superior vena cava demonstrated a very definite obstruction of this vessel with the density of the paramediastinal mass present both medial and lateral to the superior vena cava. The presumptive diagnosis of bronchiogenic carcinoma with mediastinal metastasis was made and confirmed at a later date upon the discovery of an involved right axillary lymph node. (See Fig. 10.)

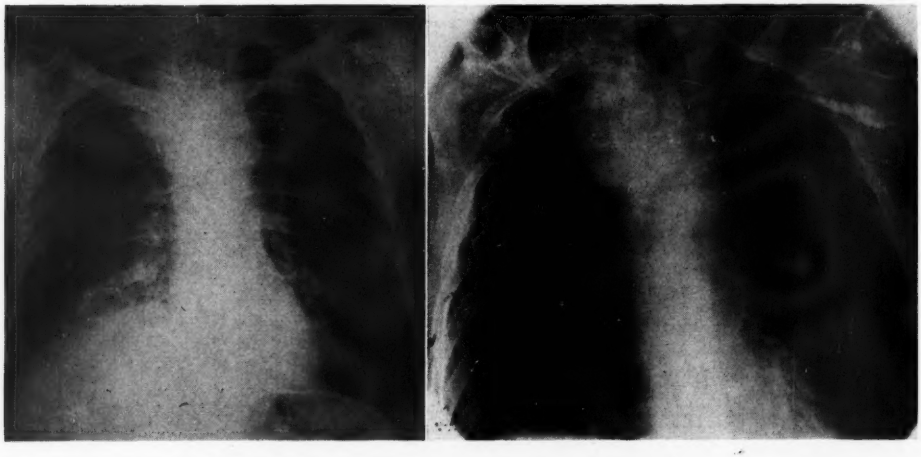


A. *B.*

Fig. 10, *A* and *B* (Mr. A. O. L.).—Right paramediastinal mass. In *B* is seen venogram showing obstruction and lateral displacement of right innominate vein.

3. *What Is the Relative Place of Angiocardiography in the Study of Mediastinal and Paramediastinal Lesions?*—Certainly a satisfactory answer to this question will not be obtained without considerable further use of this method of diagnosis. It is felt that certain basic concepts can be presented. There is frequently considerable difficulty in defining the site of origin of masses in the right superior paramediastinal region. The normal position of the right innominate and superior vena cava structures make venographic studies of considerable value in this area. It is felt to be a more uniformly applicable and less

dangerous method of differential diagnosis than the institution of an artificial pneumothorax. Unfortunately, the constant anterior position of the left innominate vein does not usually allow this procedure to be of value in left superior paramediastinal masses. There may be confusion between aneurysms of the innominate, the left common carotid, or left subclavian arteries, with tumors of lymphatic origin or thyroid. It has been our experience on several occasions to change the presumptive diagnosis of lymphoma in the presence of a right paratracheal mass to that of syphilitic aneurysm of the innominate artery. A similar experience has occurred in the presence of a syphilitic aneurysm of the left common carotid artery when the presumptive diagnosis was substernal thyroid adenoma. There is little question of the value of angiocardiography of lesions of the middle mediastinum, which, by definition, is that area of the mediastinum consisting of the pericardium and its contents. A valuable supplementary method of diagnosis in lesions of this area consists in the use of cardiac catheterization. In lesions of the anterior and posterior mediastinum, angiography may be of relatively little value unless it is required for the differential diagnosis between intrinsic and transmitted pulsation. In the study of masses of the lower portions of the anterior and posterior mediastinum, the use of pneumoperitoneum and pneumothorax is usually adequate for diagnosis and should be utilized before the use of angiocardiography.



A. *B.*

Fig. 11, *A* and *B* (Mr. W. B.).—Right paratracheal mass with associated atelectasis of right lower lobe. Angiocardiogram shows this to be a large aneurysm of the innominate artery.

Unfortunately we cannot present any evidence of the value of visualization of the inferior vena cava which has been described by other authors.[6, 7, 8] In retrospect there are at least three instances in our experience in which this method could have been of considerable value. In one patient with a chondrosarcoma of the chest wall, presenting x-ray evidence of projection toward the area of the inferior vena cava, exploratory operation revealed definite evidence of invasion of this structure, with marked displacement. A second instance of

unusual aneurysm of the heart wall with marked displacement of this structure could also have been considered. A third potential instance of an incarcerated hernia of the foramen of Morgagni, with compression of this vessel, might also have yielded diagnostic information upon the use of angiocardiogram.

The subject of the various methods of diagnosis of mediastinal tumors is contained in other publications by one of us (O.A.A.).[9] Cases felt to demonstrate points brought out in this discussion are illustrated by the following reports.

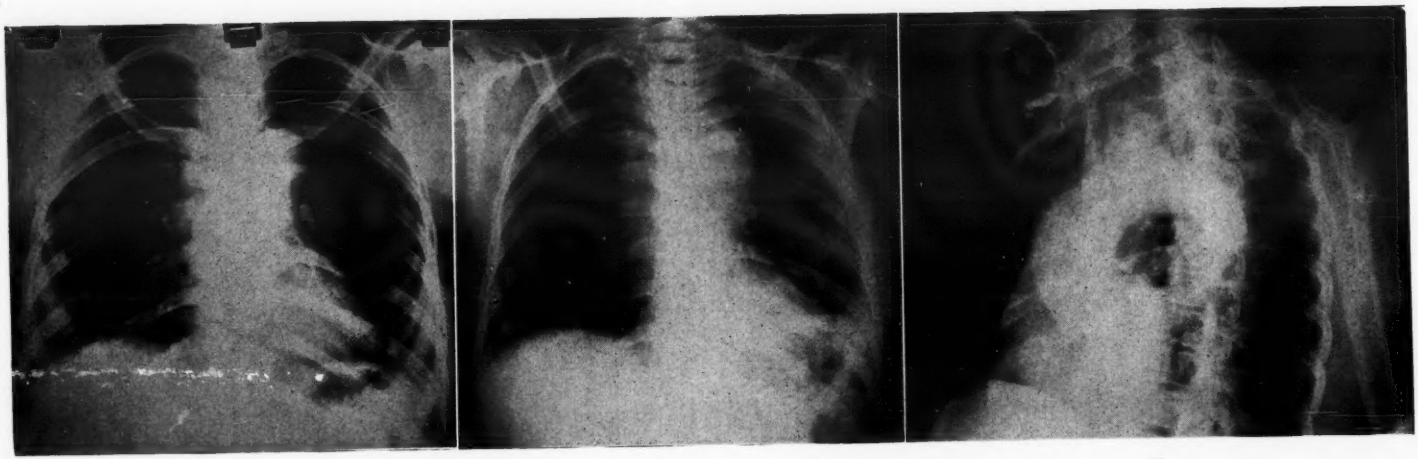
CASE 1.—W. B., a colored man 47 years of age, was admitted because of fever, pain in the right lower part of the chest, and cough. The patient had clinical findings of pneumonic consolidation of the right lower lobe, but x-ray study also showed the presence of a large right superior paramediastinal mass. There was also evidence of some partial obstruction in the right upper and middle lobes. Fluoroscopic examination failed to reveal any evidence of intrinsic or transmitted pulsation in the right paramediastinal shadow. Further x-ray examinations were felt to be suggestive that this lesion was a lymphoma. The presence of a positive Wassermann test and a degree of partial obstruction to the right upper lobe were felt by others to be suggestive that the lesion was aneurysmal in character. The presence of a syphilitic aneurysm of the innominate artery was proved by the use of angiocardiography. This was confirmed at operation when the lesion was treated with the application of Polythene cellophane. (See Fig. 11.)

CASE 2.—G. D., a 33-year-old colored woman, was admitted to the hospital because of intractable cough. X-ray examination revealed the presence of a superior mediastinal mass, rather centrally placed, with some deviation of the trachea to the right. The basal metabolic rate was plus 60, and there were several clinical findings suggestive of thyrotoxicosis. The type of distortion of the trachea, the presence of partial obstruction throughout the pulmonary tree, and the presence of a positive Wassermann test seemed to suggest the possibility of aneurysmal disease. This could be confirmed only by the use of angiocardiogram, which demonstrated the presence of a syphilitic aneurysm of the left common carotid artery. This patient was treated by bilateral decompression procedures, and subsequent Polythene cellophane application to the aneurysm. Following decompression procedures, the suggestive evidence of thyrotoxicosis disappeared. (See Fig. 12.)

CASE 3.—Mrs. R. S. W. was admitted with the primary complaint of marked exertional dyspnea. The patient had a slight prominence with suggestive pulsation in the third left anterior interspace. She showed severe evidence of dyspnea and cyanosis on very mild effort despite the presence of a vital capacity of 4.2 liters. Fluoroscopic examination revealed the presence of a large mass in the left paramediastinal area, with apparent intrinsic pulsations. Angiocardiographic studies proved the presence of a massive aneurysm of the main pulmonary artery. This lesion showed rapid progressive enlargement and was treated by the application of Polythene cellophane. Ventricular fibrillation occurred during the postoperative period, which was fatal, and autopsy examination revealed an extremely diffuse arteriolosclerosis of the entire pulmonary arterial bed. This patient has been reported in detail elsewhere. (See Fig. 13.)

CASE 4.—Mrs. J. A., a 67-year-old white woman, was discovered to have a large mass in the right cardiophrenic angle. The diagnosis of this lesion was very readily proved by the institution of a pneumoperitoneum, revealing the filling of a large hernial sac surrounding an omental hernia entering the thorax through the foramen of Morgagni. (See Fig. 14.)

4. *What Are the Special Technical Problems Associated With Venography and Angiocardiography in the Different Types of Mediastinal Lesions Encountered?*—The complete answer to this question is not considered applicable in a paper of this type and would more appropriately occur in one of the jour-



A. *B.* *C.*

Fig. 12 (Mrs. G. D.).—Superior mediastinal mass. *A*, *B*, Posteroanterior views show before and after decompression of the chest. *C*, Angiocardiogram shows filling of the mass from the aorta.

nals of roentgenology. We do feel that it is important to mention that it is frequently necessary to obtain angiocardiographic studies in different positions. In some cases it would appear to be of value to obtain a venogram in the lateral

A.

B.

Fig. 13 (Miss R. S. W.).—*A*, Posteroanterior view shows mass, middle mediastinum. *B*, Angiocardiogram shows massive aneurysm of the left pulmonary artery.

position and a subsequent angiocardiogram in the anteroposterior or one of the oblique positions. It seems to be particularly valuable in lesions which are known to distort the esophagus, to obtain the angiocardiogram with contrast media also present in the esophagus. In certain instances the dilution factor

may be sufficient that a satisfactory visualization of the aorta and its major branches cannot be obtained by the classical method of injection of the Diodrast media into a vein. In such conditions it would be advisable to carry out one of the various methods of direct arterial injection of the contrast media. This can

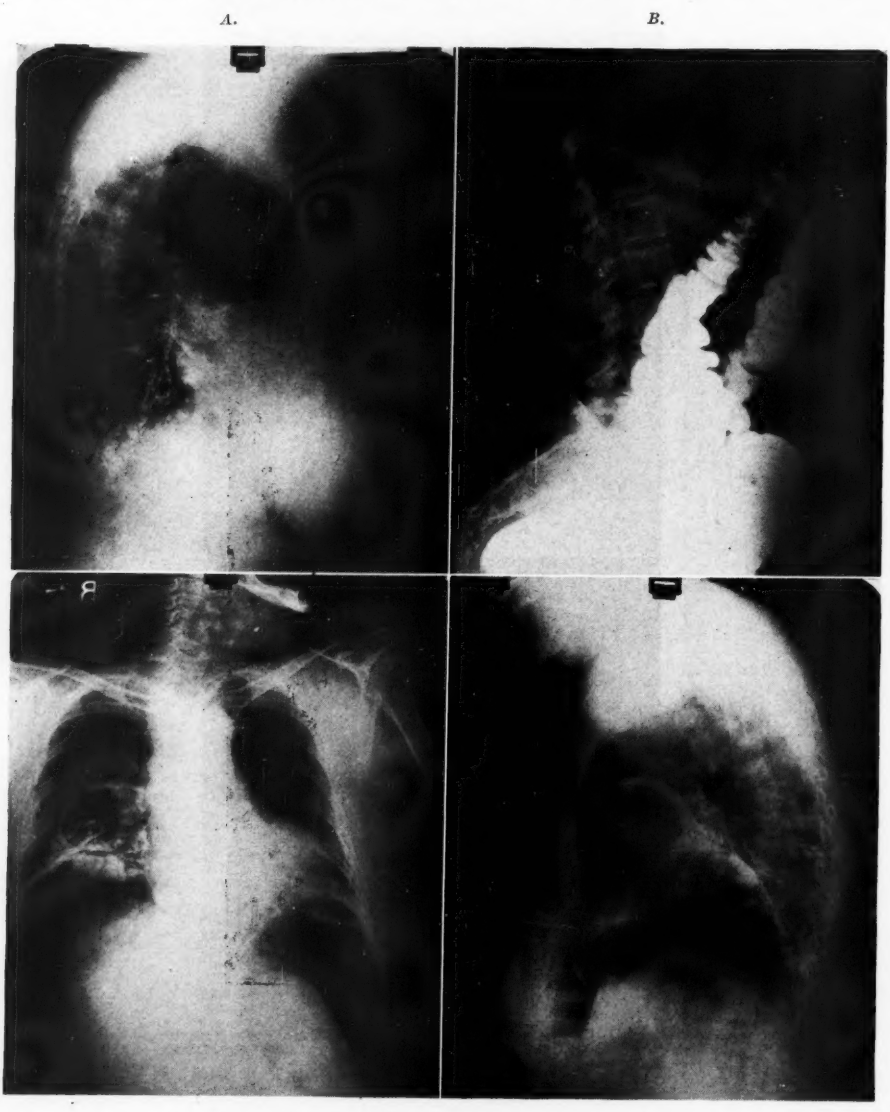


Fig. 14, *A* to *D* (Mrs. J. A.).—Foramen of Morgagni hernia showing two diagnostic features: *B*, barium study of colon shows inverted "V" formation. Pneumoperitoneum showing filling of hernia sac above diaphragm.

be done by catheterization of the radial artery, or by direct injection into one of the carotid arteries for retrograde injection. It is felt to be of utmost importance that a very careful fluoroscopic examination and review of the various directional x-ray examinations should be carried out first in order to determine the proper position for angiocardiography. In some instances angiocardiography may be repeated at the same sitting in different positions.

It is also felt that lesions appearing in the superior mediastinum on the left should first be examined by injection of the contrast medium into the antecubital vein of the left arm. In comparable fashion, right superior mediastinal lesions would require use of the right antecubital vein. In some instances this will reveal a complete obstruction of this structure and satisfactory angiocardiography can be carried out only by use of the contralateral arm. It is also suggested that in some instances it could be of value to do concomitant bilateral injection of the contrast substance. Exemplary patients are presented as follows.

CASE 1.—Mrs. R. T., who has been described previously as showing deviation of the right innominate vein in relation to a substernal thyroid adenoma, should also have been studied in the lateral projection to determine the effect upon the superior vena cava. This would have required injection of the contrast media into the opposite arm from that which was used. (Fig. 8.)

CASE 2.—J. P., a 51-year-old white man, was admitted with symptoms of circulatory disturbances in the right arm in association with symptoms of tracheal compression. The initial attempts at angiocardiography were unsuccessful due to the complete obstruction of the vein. It was necessary to inject the left antecubital vein in order to obtain a satisfactory angiocardiogram. There was just sufficient contrast medium present to allow the diagnosis of aneurysm of the innominate artery, and a more satisfactory visualization could have been obtained by retrograde arterial angiocardiography. (See Fig. 15.)

CASE 3.—J. H., a 46-year-old colored man, was admitted with the main complaint of esophageal obstruction, weight loss, and thoracic pain. X-ray studies revealed the presence of a large mass displacing the esophagus posteriorly in its mid-portion. The lesion did not appear to pulsate on fluoroscopic examination, and appeared to be of uniform density on the study of the lateral x-ray projection. Angiocardiographic study was carried out with concomitant visualization of the esophagus with barium. The angiocardiogram seemed to show that the lesion was not of vascular origin. Unfortunately the patient suffered a rapid downhill course, and post-mortem examination was not permitted.

5. *Of What Value Can Angiocardiography Be in the Study of Certain Primary Pulmonary Lesions, in Particular, Pulmonary Neoplasm?*—This topic has been mentioned in some detail elsewhere, with mention that a rather large percentage of patients having primary carcinoma of the lung show a definite disturbance of the vascular pattern of the pulmonary tree.[11] It is also known that a certain percentage of patients will not show abnormality of the vascular bed in the presence of pulmonary malignancy, and it will involve, therefore, a very considerable number of cases to determine the exact value of this procedure in relation to carcinoma of the lung.

In recent months we have tried to institute this procedure as a routine part of the diagnostic work-up of probable pulmonary malignancy patients. It has been used in fourteen instances, and in twelve patients definite evidence of vascu-

lar abnormality could be demonstrated. In four instances a demonstrable distortion of the pattern of the superior vena cava was present. In seven patients unquestioned abnormality of the pulmonary arterial system could be demonstrated. These all showed varying degrees of obstruction of the nutrient artery to the involved area of the lung. It was of most demonstrative value in lesions

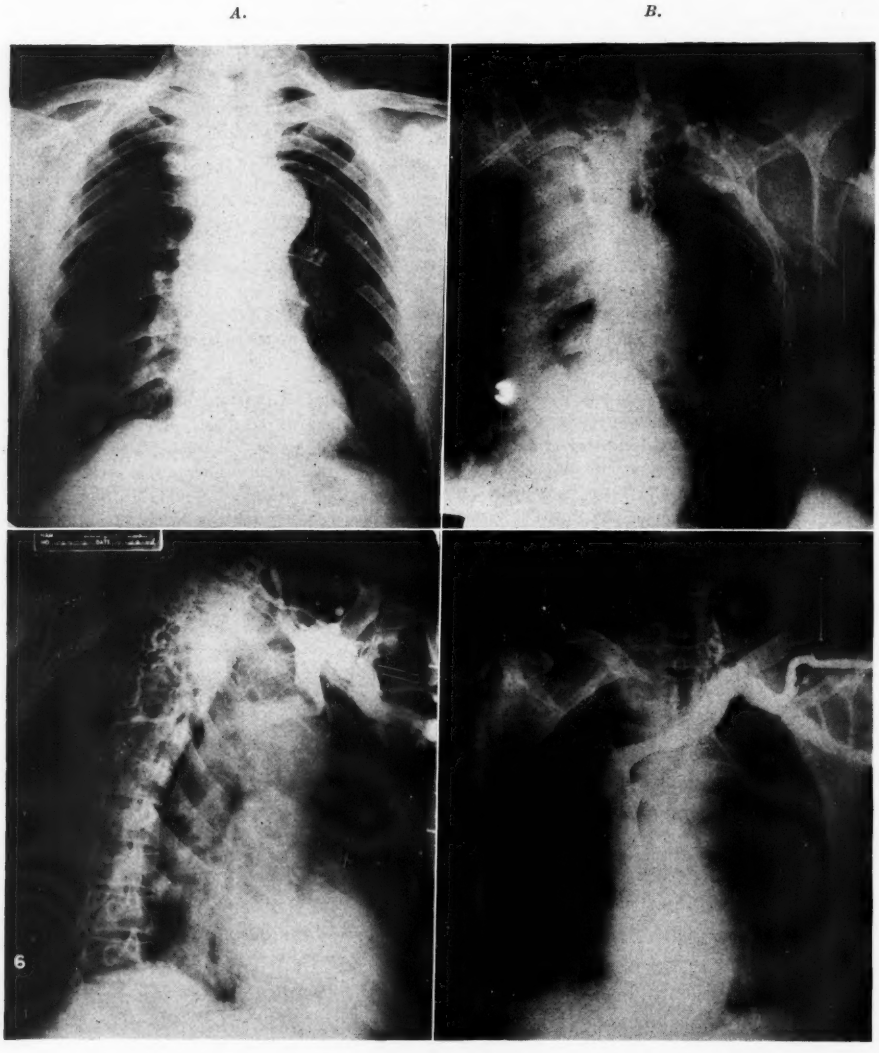


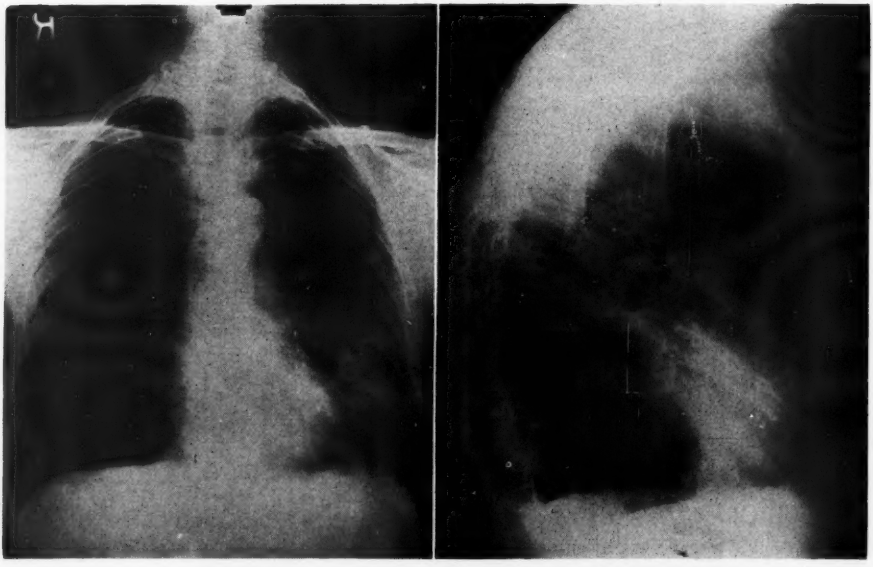
Fig. 15, *A* to *D* (Mr. J. P.).—D, Angiocardiogram on right shows obstruction to innominate vein with considerable retrograde flow into lesser radicles. *C*, Repeat gave adequate filling.

A. *B.* *C.*

Fig. 16. *A* to *C* (Mr. J. H.).—Mass anterior to esophagus displacing esophagus posteriorly. *A*, Angiocardiogram study with contrast barium to esophagus shows the lesion to be extravascular.

of the upper lobe beyond the range of the bronchoscope, and which we had not been able to visualize satisfactorily by Lipiodol studies. It is felt to be a potential valuable adjunct in the study of upper lobe tumors.

It has also been our experience recently to do exploration in a patient with carcinoma of the lingual segment associated with massive pleural effusion as his earliest symptoms. The pleural fluid was clear, and at operation the lesion was found to be entirely confined to the origin of the lingual bronchus, and the effusion secondary to infarction of the entire lingula due to malignant thrombosis. Certainly, in retrospect, angiocardiographic study might have been of considerable value in this patient. (See Fig. 17.)



A. *B.*

Fig. 17, *A* and B (Mr. S. B.).—Carcinoma of the lingual orifice with infarction of lingual artery. Angiocardiographic studies would have been of considerable value in this patient.

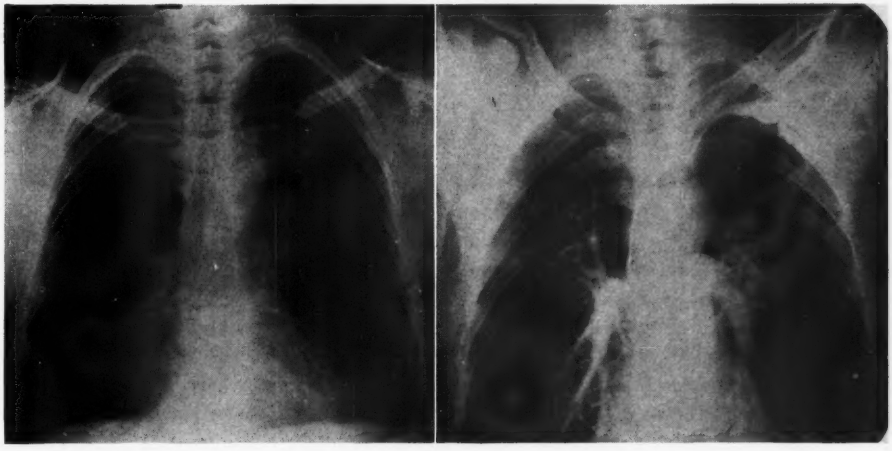
On three occasions this procedure has been carried out in the presence of nonmalignant pulmonary disease. In one case, a chronic interstitial pneumonitis of the median and dorsal basilar segments of the right lower lobe was found not to be associated with any distortion of the pulmonary vascular pattern. The second instance of suppurative disease, chronic, of the right upper lobe was found to be associated with a normal pulmonary vascular pattern. A third instance of a left paramediastinal mass due to chronic atelectasis of the left upper lobe was found to be associated with considerable distortion of the vascular pattern, but not of the obstructive type.

The use of angiocardiography in patients with advanced pulmonary emphysema to the degree which is now described as "vanishing lung disease" has

not become common clinical practice because of apparent inherent dangers in this type of patient. Robb and Steinberg[12, 13] have described the pattern noted in one patient as "advanced pulmonary emphysema and fibrosis." We have carried out studies in this clinic in post-mortem specimens obtained from patients with "vanishing lung syndrome," and do feel that there is a specific pattern associated with this lesion. In the gross examination of these lungs, one is struck by the apparent disappearance of all the finer elements such as the alveoli, bronchioles, and small arteries and arterioles. Injection of the bronchi of these lungs confirms this impression, and similarly injection of the vascular tree has shown that the areas of greatest disappearance of the smaller vascular radicles is associated with the areas of major disappearance of other pulmonary structures. The application of this form of investigation in this type of patient will need to await further clinical and experimental studies.

Case histories exemplifying these statements are presented here.

CASE 1.—W. N., a 42-year-old colored man, was admitted with symptoms compatible with carcinoma of the lung. X-ray examination showed evidence of obstruction in the region of the left upper lobe. Bronchoscopic examination proved noncontributory. Lipiodol studies were repeatedly unsatisfactory for visualization of this area. Angiocardiography showed a complete obstruction of the apical segment of the left upper lobe pulmonary artery. (See Fig. 18.)

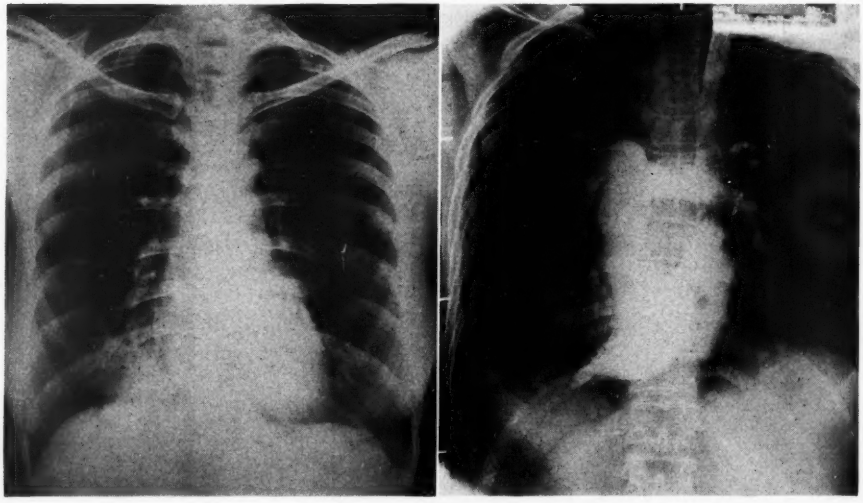


A. *B.*

Fig. 18, *A* and *B* (Mr. W. N.).—Note complete blockage of apical branch of left pulmonary artery in region of lesion.

CASE 2.—Miss L. C., a 48-year-old white woman, was admitted with the complaint of repeated severe hemorrhages with subjective localization of the point of origin to the right lower lobe. X-ray examination showed infiltration in this area with apparent partial obstruction. Bronchoscopic examination proved noncontributory. Angiocardiographic studies revealed what appeared to be a completely normal pulmonary arterial vascular pattern. At operation this lesion was resected by segmental resection, and proved to be a chronic interstitial pneumonitis. (See Fig. 19.)

CASE 3.—M. G., a 43-year-old white man, was admitted with the complaints of progressively severe dyspnea, and marked limitation of activity. He had long suffered from intractable bronchial asthma. Following intensive study the patient was operated upon. Massive pneumatoceles were removed or plicated in the right lung, and a right pulmonary plexectomy was performed. The patient progressed very well following this procedure, and five months later returned for plexectomy operation on the left lung because of persistence of bronchial asthma. The patient suffered acute ventricular fibrillation ten days following the operative procedure, and at autopsy examination the lungs showed evidence of advanced pulmonary emphysema to the degree known as "vanishing lung disease." The lungs were studied by means of Lipiodol special contrast media to visualize the bronchial and vascular tree.



A. *B.*

Fig. 19, *A* and *B* (Miss L. C.).—Mass, right lower lobe, which was proved to be a chronic interstitial pneumonitis. *B*, Angiocardiogram entirely normal.

In review of cases seen with lesions of the mediastinum and paramediastinal areas, angiocardiography has been of diagnostic value in thirteen instances. It has been of considerable aid in diagnosis and therapy in ten cases, giving a total of twenty-three cases in which definite help was obtained from this method of investigation. It was of questionable value in only seven cases, lending indirect information in these cases. This information cannot be considered of great importance until more cases have been worked up. In six cases the procedure proved of no value at all.

SUMMARY AND CONCLUSIONS

We have reviewed thirty-six patients in whom mediastinal or paramediastinal masses were studied by means of venography or angiocardiography, or a combination of both. The series is obviously too small to derive any permanent conclusions. Certain impressions have been obtained and discussed. The subject has been discussed primarily in relation to five fundamental questions. Patients

exemplifying factors involved in each of these categories have been presented. An over-all chart summarizing the findings in all forty cases is included. The following conclusions are suggested.

1. There is a definite place for the use of venography and angiocardiography in the study of mediastinal and paramediastinal masses.

2. The angiocardiogram provides the most valuable method of differentiation between vascular and avascular lesions situated in this area.

3. This method of investigation has a definite but limited use in the study of the nonvascular tumors of the mediastinum. Its primary function in this situation is to aid in a better localization of the lesion and a better outline of its borders.

4. The relative value of angiocardiography as a method of the study of these lesions varies considerably with the area of the mediastinum involved. It is of absolute necessity in the study of all lesions of the middle mediastinum. It is of almost equal importance in the study of lesions of the superior mediastinum, especially if they are located in the right paramediastinal area.

5. Certain special technical problems associated with each type of lesion have been discussed. It is vitally necessary that careful fluoroscopic examination and multiple x-ray projection studies be carried out before determining the particular positions and combinations of angiographic methods to be utilized in the individual study of a specific case.

6. It is felt that angiocardiography can be of specific value as an aid in the study of bronchiogenic carcinoma, in particular, in lesions of the upper lobes. A specific distortion of the vascular pattern is felt to occur in patients with advance pulmonary emphysema of the type known as "vanishing lung disease."

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DISCUSSION ON "ANGIOCARDIOGRAPHY AND AORTOGRAPHY IN THE DIAGNOSIS OF CONGENITAL CARDIOVASCULAR LESIONS" BY DR. THOMAS H. BURFORD, DR. MERL J. CARSON, AND DR. WENDELL G. SCOTT* AND "THE ROLE OF ANGIOCARDIOGRAPHY AND VENOGRAPHY IN MEDIASTINAL AND PARAMEDIASTINAL LESIONS" BY DR. OSLER A. ABBOTT, DR. WILLIAM A. HOPKINS, AND DR. TED F. LEIGH

DISCUSSION

DR. G. H. HUMPHREYS, New York.—I should like to start by congratulating Dr. Burford and his group on these beautiful studies. It is not easy to get such fine demonstrations of these lesions, as anyone who has tried to work out a system knows. The group at Presbyterian Hospital, Dr. Deterling, and Dr. Ball have been working on this problem for some years, and we have succeeded in getting what we consider successful intravenous angiocardiographies in congenital lesions which, in some cases, seemed to us so satisfactory that the more elaborate retrograde injection seems unnecessary. I should like to show a few slides to demonstrate what I mean. (Slides.)

This is the ordinary type of adult coarctation and shows, as Dr. Burford brought out, the lesion here. The next shows a child in which the coarctation at this point is shown more clearly through the more rapid injection of dye. This we have found to be the most important factor, and as you see there is a sufficient segment to assure an adequate anastomosis. The next slide shows a tetralogy of Fallot taken in the anteroposterior position; the needle is large and is placed in the external jugular vein, the dye entering the heart goes out the aorta immediately, demonstrating the overriding aorta; the pulmonary arteries are well shown in the original film, and a small constricted pulmonary artery can be seen, thus demonstrating clearly that the lesion is operable.

The next slide shows a more complicated lesion. I should like to take small issue with Dr. Burford on these complicated anomalies of the great vessels. I think they show better frequently by intervenous methods. This was a cyanotic girl who, by cardiac catheterization, gave many evidences of poor pulmonary flow and so presumably was a case for operation. However, the ordinary x-ray film showed some anomaly at the aortic arch and, as we can see here, the arch bifurcates and this is in fact a double aortic arch. It is seen to split here and here; there is a double shadow, one passing downward and entering the pulmonary artery and another passing downward as the descending aorta. On the lateral view, less clear on the slide than on the film, there is quite clearly a coarctation in the right side of the aortic arch; that is, the right arch of the double arch is coarcted. In other words, this patient is demonstrated by these films to have not only an intraventricular septal defect and overriding aorta, but also a double aortic arch and a patent ductus arteriosus, as well as coarctation of the aorta. She has all four of the lesions which are operable, but it adds up to an inoperable situation since all the blood to the lung is entering through the patent ductus arteriosus. You will note the left lung has dye in it, the right has none; a few seconds later the right lung filled from collateral vessels. She has, therefore, no right pulmonary artery for anastomosis, and operation on the left side would probably be fatal since that is the lung which is supplying most oxygen.

DR. ANDRE COURNAND, New York.—After the remarkable demonstration given by Dr. Burford, I hesitate to take issue with one statement he made concerning catheterization of the heart in young children and infants. At Bellevue Hospital, in cooperation with Dr. Himmelstein and Dr. Janet Baldwin, we have catheterized infants as young as 5 months and weighing as little as 8 pounds. The technique used is somewhat different from that employed in adults. The catheter is introduced into the saphenous vein, which has to be ligated at the end of the catheterization. A special small catheter, No. 6, with a very small bend near the tip has been devised for such cases, which permits easy access to the right ventricle and the pulmonary artery without creating a big loop in the right auricle and superior vena cava.

*See page 860 for article by Burford, Carson, and Scott.

In one case it was demonstrated today for the first time that it is possible to visualize a patent ductus arteriosus with contrast medium. This is rather important because a differential diagnosis between an aortic septal defect and a patent ductus arteriosus is impossible with the catheterization technique.

I wonder whether, as stated by Dr. Burford, it is possible to distinguish a nonfunctioning right ventricle from a single auricle associated with a single ventricle.

Dr. Burford should certainly be complimented upon the fine demonstration of the results obtained by combining the conventional venous angiocardiography with the aortagrams.

DR. ARTHUR S. W. TOUROFF, New York.—I would like to join with Dr. Humphreys and Dr. Cournand in congratulating Dr. Burford and his associates on their beautiful demonstration of the value of visualizing the great vessels in the presence of congenital cardiovascular abnormalities. At The Mount Sinai Hospital in New York, we have been most fortunate in having the services of Dr. Marcy Sussman, one of the pioneers in angiocardiography in America. We also are greatly indebted to our physicist, Dr. Serge Feitelberg, for making many improvements in the apparatus which we use. In the differential diagnosis of certain lesions, it is important to take films at a rapid rate. Accordingly, our apparatus is able to make exposures at a rate of more than two per second. (Slides.) Our apparatus consists essentially of a battery of lead-backed cassettes which are held, one in front of the other, in a magazine. As each exposure is made, the front cassette drops into a hopper, thereby immediately leaving an unexposed cassette ready for the next exposure. The magazine holds fifteen cassettes which can be exposed in seven seconds if desired. An important component of our apparatus is the "amplifier-synchronizer." This permits us to synchronize the taking of films with the electrocardiograph. Through the medium of a series of twenty-two switches, which may be pre-set as desired, exposures also can be made automatically at any desired interval. Thus, the apparatus can be operated manually, or the switches may be set to take films at predetermined intervals, or finally the apparatus may be controlled by the electrocardiograph to take films in any desired phase of the cardiac cycle. The latter at times is important because, under certain circumstances, great errors in interpretation may be made. I would like to demonstrate how such an error may occur. (Slide.) Here is a film taken four one-hundredths second after the peak of the R wave. You see the dye coming down the superior vena cava to enter the right atrium. The exposure has been made at the beginning of systole of the right ventricle. The pulmonic valve and pulmonary artery are outlined, and the ventricle is filled with dye. (Slide.)

This second film is taken twenty-five one-hundredths second after the peak of the R wave. This is the end of systole of the right ventricle. You will note that, in this view, the right ventricle is practically empty. However, the pulmonary artery is filled with dye. If, by accident, one happened to make the exposure at this particular time without knowing what came before, one might be led to the conclusion that the patient was suffering from pulmonic stenosis. Incidentally, this film illustrates the fact that the left pulmonary artery normally rises to a higher level than the right. (Slide.)

This third film is taken precisely two heartbeats after the filling of the right atrium. The dye fills the right auricle, the right ventricle, the pulmonary artery, the left auricle, the left ventricle, and the aorta. Accordingly, the diagnosis of a septal defect is obvious. The common carotid with its branches, the external and internal carotid, are well visualized. At this point, the right subclavian is seen, and here are the left carotid and left subclavian arteries.

Our x-ray department has done more than 2,000 angiocardiograms without a death. We attribute this to the fact that every patient is tested very carefully for sensitivity to iodine. A test dose of 1 c.c. intravenously is given to every adult; every child receives 0.5 c.c. intravenously. An untoward reaction to these test doses eliminates the patient from further study by means of angiocardiography. Practically every patient has some reaction during angiocardiography, although these reactions usually are mild. Weakness, pallor, sweating, and a feeling of warmth are common. However, we have never had a death, nor have we observed any serious complications following the procedure.

Those who are interested will find a description of the apparatus which we employ in a forthcoming publication.*

DR. OSLER ABBOTT, Atlanta, Ga.—I wish to bring to your attention the role of the ballistocardiogram in the study of certain congenital heart lesions. (Slides.) This is the instrument which is used, and its fundamental use, of course, is determination of cardiac index and cardiac output. The thing that is interesting is the changes that occur in coarctation, and that there is a specific wave with this machine in two diseases, coarctation of the aorta and primary thrombosis of the aorta. The fundamental change is seen in the K wave. The K wave in coarctation of the aorta preoperatively, shows the notching of the descending limb in that wave. The I wave is also of interest, and is mainly an indication of an overactive heart, and is increased in certain lesions. The postoperative tracing on a patient in whom a direct aortic end-to-end anastomosis was possible, and in whom there was a large distal aorta, showed essentially complete conversion of the K wave to normal.

In another patient the K wave was even more distorted with a low fall and very jagged descent, and there was a very marked increase in the I wave in the descending limb. This patient was operated upon in the early group by an end-to-side shunt, using the subclavian artery to the aortic wall, and it showed in the postoperative period that the conversion of the K wave was in no way comparable to the satisfactory conversion in the preceding patient.

Here is the tracing made on another patient with the preoperative K wave markedly flattened, and in the postoperative period the flattening was corrected, particularly in the height of the limb of the K wave, better than the preceding one; the notching persisted because of severe hypoplasia of the entire descending thoracic aorta, noted at the time of end-to-end aortic anastomosis.

The same thing can be seen in another patient in whom there was a marked degree of aortic insufficiency, showing the marked enlargement of the I wave in the preoperative stage, and this shows the postoperative change in the K wave which we feel is satisfactory, though a little limited because of the fact that the aorta was rather smaller than normal, but the I wave is not changed because the insufficiency, of course, has not been radically corrected.

We feel this is an additional method not only of making diagnosis of coarctation of the aorta but also evaluating postoperative results in different types of methods used.

DR. H. BRODIE STEPHENS, San Francisco.—I think a word of caution should be given at this time; perhaps Dr. Burford has it in his original manuscript. It is very important to remember in retrograde carotid arteriograms to block off completely the distal carotid, particularly when a 70 per cent Diodrast solution is being utilized. Neurosurgeons, of course, have given up this solution in their visualization of the cerebral vessels. Only recently, in one of our cases, unfortunately the distal carotid was not obliterated, and the patient developed hemiplegia on the right side when we used the left common carotid. Fortunately, he recovered by stellate ganglion block on three different occasions. But this is a very definite danger in retrograde arteriograms, and we now have adopted the technique of always completely obliterating the distal carotid by the use of a bulldog clamp.

DR. E. E. AVERY, Chicago.—Dr. Burford and his associates are to be complimented for developing their excellent techniques and mechanical devices to improve this procedure. At Hines Hospital, we have carried out this dye visualization in well over fifty cases involving the differential diagnosis of mediastinal and paramediastinal masses. I would like to emphasize that this can be a relatively easy procedure which can be carried out in any hospital without expensive equipment. We use a Robb-Steinberg syringe and needle and a standard stereocassette changer to take our roentgenograms. It takes only a little practice and patience in developing the proper technique of circulation times and in taking exposures at the right intervals. Certainly in infants with cardiovascular anomalies, it is necessary to

*Medical Physics, ed. 2, edited by Dr. Otto Glaser, Chicago, 1949, The Year Book Publishers, Inc.

have rapid multiple exposures to obtain the desired information. However, in adults with mediastinal lesions, the diagnostician should be encouraged to try this procedure with the simple equipment mentioned, because it will yield n great deal of valuable information.

Where there is vascular obstruction of the mediastinum, as is illustrated in this slide, we have found the angiocardiogram of value to the surgeon. Here can be seen a massive obstruction; the subclavian is blocked, and the dye has to come in through the supreme intercostals into the azygos system. This is of value in three ways: it allows the surgeon to know better just what problems he will encounter; by knowing the major collaterals, damage to them can be avoided during exploration of the chest; if the case is inoperable, it helps the x-ray therapist to direct the roentgen bombardment to the area at which the tumor mass has occluded the vessels—in other words, he has a better idea as to the location of tumor tissue in the mediastinal shadow.

Every surgeon has at one time or another found an aneurysm at exploratory thoracotomy where he fully expected to find tumor, and vice versa. Therefore, angiocardiography should be done in mediastinal lesions because: (1) It aids in planning the approach and the procedure to be used; (2) it allows the surgeon to avoid unnecessary exploration; (3) it confirms and justifies necessary thoracotomies.

I want to re-emphasize that this valuable information can be gained in appropriate cases with a little experience and with relatively simple apparatus available in any hospital. Watchful waiting in these lesions is dangerous, and the danger of the procedure itself is by far outweighed by the valuable information which can be obtained.

DR. J. A. HELMSWORTH, Cincinnati.—The approach to n small portion of the problems covered so well by Dr. Abbott has been a method of arteriography by countercurrent catheterization of the aorta. The contrast medium, Diodrast, is injected in small volumes rapidly and with the usual simple x-ray apparatus single films are taken. (Slides.)

This shows the catheter that has been improvised. It is made of Polyethylene tubing with an outer diameter of .098 and an inner diameter of .068 and with a 15 gauge needle used as an adapter. This is a posteroanterior view of the chest of a patient with syphilis and under suspicion for aneurysm of the innominate artery. With the catheter inserted through the brachial artery at the level of the distal end of the humerus, the tip was advanced to the origin of the innominate artery and with the injection of approximately 10 c.c. of Diodrast some visualization of the small remaining lumen was obtained. The thickness of the wall of the aneurysm was outlined, as well as the vertebral artery.

This patient also had syphilis. In the posteroanterior view the location of a mass suspected of being aneurysm was outlined. With the introduction of the catheter through the brachial artery on the right, advancing the tip down into the origin of the aorta, and the injection of a small amount of Diodrast, approximately 12 c.c., it outlined the general course of the arch of the aorta and indicated in a rather vague way the outline of the related aneurysm.

This patient also was suspected of having an aneurysm of the innominate artery, and the posteroanterior view gives the reason for this. Some difficulty was encountered in advancing the catheter beyond the brachial artery and finally, when it could not be brought into the innominate or down into the origin at the aorta, 8 c.c. of Diodrast were injected, and a definite outline of an occluding process of the subclavian is shown. This is concluded to be the wall of the aneurysm in relation to the subclavian artery and occluding the same by extrinsic pressure.

This outlines in much less contrast than Dr. Burford's cases the approximate limits of the aorta, the subclavian artery, the coarcted portion of the aorta, and the distal portion of the aorta. Here can be seen the left internal mammary artery and a little further in the same direction is the right internal mammary. This vessel is the left subclavian artery with a take-off quite near the origin of the upper limit of the coarctation.

This Negro, having been shot through the right side of the neck, was thought to have an arteriovenous communication at the level of the hyoid bone; here is the catheter through

the brachial artery. The Diodrast demonstrates very well the vertebral artery and the ascending cervical artery. This shows filling of the common carotid, the aneurysmal sac, and incomplete filling of the venous channels involved in the same process.

DR. HAROLD NEUHOF, New York.—I believe we are all equally appreciative of Dr. Abbott's presentation of this subject, the first presentation, I think, which has been made before this Society, of an extremely useful method of diagnosis in a great variety of intrathoracic conditions. His study, as you have seen, is truly encyclopedic, and you can also see the innumerable possibilities which are opened up for us all in further development of this work.

He has referred to some of our own work, and that has been particularly concerned with, first, the effort to demonstrate the influence of intrathoracic lesions which are mediastinal on the pattern of superior vena cava and innominate veins and pulmonary artery, and not in so far as intracardiac patterns are concerned; and second, the effect on the pattern of the pulmonary artery of lesions within the lung. It would go too far afield to refer to our various experiences which have, in a general way, been along the lines to which Dr. Abbott has referred this afternoon. In a recent publication in *Surgery* we reviewed our experiences. In so far as visualization in connection with mediastinal lesions is concerned, I would say that, according to our experience, a negative angiogram, that is, a normal pattern of the branches of the pulmonary artery, is often as interesting and significant as a positive one. I hope Dr. Touroff will show the films of a patient who had an enormous shadow chiefly in the anterior mediastinum, which, however, did not indent at all the pattern of the contrasted visualization of the heart and great vessels. The lesion at operation proved to be a thymoma of soft consistency. Most surgical mediastinal lesions are not related to the great vessels, and therefore, according to our experience, the contrast visualization of the great vessels has been one of a negative nature. Indeed a positive film, that is a distortion or block of one of the great vessels points to a surgically irremediable lesion in my experience.

Concerning pulmonary lesions, which I should like to discuss specifically, Dr. Abbott has pointed out the block of a branch of a pulmonary artery in some cases, which has confirmed for him, and I think entirely properly so, the diagnosis of cancer of the lung. We are of the opinion, according to our experiences which have been reported, that an angiogram or rather a pulmonary arteriograph which demonstrates a block of either the main trunk or a major branch is truly a pathognomonic sign of cancer of the lung. That is, of course, a dangerous thing to say, but it has been so in our experience up to the present. The important point is that this does not at all establish that such a lesion is inoperable. Steinberg, a master in the field of pulmonary angiography, some months ago referred to a number of cases in which he noted this blockade, and it was his opinion and his experience that these blocks accompany an inoperable condition. However, in a discussion of the matter, he agreed that it was perhaps the type of material with which he came in contact which lead to this view. We have seen upon a number of occasions the diagnosis definitely established of cancer of the lung, which could not otherwise have been established by angiograms. Since we all agree that we are no longer in the stage when we say let us explore and find out the nature of the lesion, if we can diagnose the lesion prior to operation I think we should bear in mind not only the possibility but the probability of specific aid in diagnosis by this method which, I should like to emphasize as Dr. Abbott has already done, is entirely free from danger when performed under proper auspices.

DR. ROLLINS HANLON, Baltimore.—I should like to emphasize the word of caution which Dr. Burford expressed in his excellent presentation. We have not been as fortunate as Dr. Sussman and his collaborators in their tremendous experience of some 2,000 cases. The experience of our radiologic and surgical departments comprises more than 600 injections in over 300 patients with congenital cardiovascular disease, predominantly of the cyanotic type. While these are admittedly substandard risk patients, the fact that there have been three deaths in our series leads us to feel that the procedure should not be used indiscriminately and without serious consideration as to the benefits to be obtained by this method of examination. All of our patients received an intravenous test dose of iodo-

pyracet; it is obvious that a negative reaction to such a test does not rule out the possibility of a fatal outcome. One should not use iodopyracet injection unless he feels that the diagnostic requirements warrant the not inconsiderable danger in these substandard risk patients.

I should like to show several slides illustrating a modification of our standard technique as recently employed in cases of coarctation of the aorta. In conjunction with Dr. Cooley and Dr. Sloan of our radiology department, Dr. Harry Muller has incised the radial artery below the elbow and passed a catheter to the vicinity of the aortic coarctation. This permits better visualization of the vessels about the coarctation, while employing considerably smaller amounts of iodopyracet than is usually given by the intravenous method. The first slide shows an excellent view of an aortic coarctation after injection of 50 c.c. of iodopyracet through an antecubital vein. The clarity of detail is much better than we usually obtain by this technique. In contrast, the next slide represents the film obtained after injection of only 20 c.c. of the dye through a catheter with its tip lying near the coarctation; the detailed anatomy of the region is seen much more clearly. In the next slide can be seen a demonstration of patent ductus arteriosus in conjunction with coarctation, and in the final slide there is demonstrated an aneurysm of the intercostal artery just below a coarctation. In this latter instance the additional surgical procedure and the increased risk incident to aortic catheterization seemed justified by the additional information which was of great value to the surgeon.

Angiocardiography has been used in all but a few of the forty-six patients with coarctation of the aorta operated upon by Dr. Blalock and his associates. Recently, injection through an inlying aortic catheter has replaced simple intravenous injection in patients suspected of coarctation. It is to be emphasized, however, that intravenous injection is still used as the customary technique in most other instances, especially in the poor-risk, cyanotic patient.

DR. CARSON.—Being a pediatrician I rather feel that I should apologize for appearing before this meeting. It is a great privilege, however, and my only excuse is that I am a part of a team, with the thoracic surgeons, in trying to work out these congenital malformations of the heart. There are a number of points I would like to emphasize in detail. The first refers to Dr. Cournand's question about the differentiation of the nonfunctioning right ventricle, as shown by angiocardiography, as differentiated from the picture of a single great auricle and a single ventricle. Again I must apologize for omitting the first slide in the series Dr. Burford showed. This was omitted for the sake of brevity. However, on the first picture obtained on that patient, taken about two-thirds second before, one could see very definite filling of the right auricle, with no Diodrast being seen in the ventricle. There was a very small amount of Diodrast in the left auricle at that time. In the following plate, as you saw, could be noticed the right auricle, the left auricle, and the left ventricle, with no right ventricle being visualized.

Again I wish to emphasize that this is another laboratory procedure. One should not attempt to make it the only laboratory procedure or method of examination used. We use it in conjunction with the other studies, and these other studies would also help reveal the difference between the two conditions.

I would like to comment on Dr. Touroff's question regarding test doses of Diodrast. We feel this is extremely important. We abandoned the intradermal testing for sensitivity to Diodrast because we obtained so many false positive reactions. In every patient on whom we do angiograms or arteriograms we inject intravenously, as Dr. Touroff does, a small amount of Diodrast before the procedure is undertaken. We also make sure of good renal function by doing a nonprotein nitrogen test before the angiogram is done.

Dr. Stephens brought up the question of obliteration of the left common carotid artery cephalad to the point of injection. That is always done; the cannula is inserted into the left common carotid artery and a ligature is tied around the cannula so there is no possibility for the Diodrast to go directly up into the brain.

I certainly would agree wholeheartedly with Dr. Hanlon's position as just stated and, in contrast to Dr. Avery's remarks, I really do not feel that this is a procedure to be

undertaken lightly. We have seen a number of reactions, particularly in the very cyanotic group of children who are in poor condition at the beginning. We feel that if other measures will give a definite diagnosis, this method of examination should be avoided. We do not feel it is an easy procedure and feel that it has very definite dangers. When it is definitely indicated, however, we feel that one should not hesitate to use it.

In conclusion, there is one further point I would like to make. In our group we feel there is a very definite challenge to the thoracic surgeons and to the pediatricians, in children, during the first few weeks or months of age, who have the infantile type of coarctation of the aorta. These are the children who present themselves with the picture of cardiac failure and not the typical picture of the adult type of coarctation of the aorta. In our experience during the last two years we have had nine such patients in St. Louis Children's Hospital, and they have uniformly died within a period of one day to three or four weeks after admission. We feel this is a definite group of patients who could and should be benefited if they can be brought out of cardiac failure long enough for remedial surgery to be performed.

DR. HOPKINS.—As has been pointed out, we too feel that these patients should be chosen carefully, not only with regard to those who will benefit by the procedure but also with regard to the selection of the patients. The radiology department has done more than 700 injections at this time without a death. They have steadfastly refused to do an injection in any patient with severe coronary disease or any patient with evidence of severe bronchospasm, and also those with an extreme degree of renal disease.

We also feel that in the use of the venogram a less amount of dye may be injected, and 35 per cent dye may be used in these patients. I would like also to say that there is one other lesion in which we feel that this procedure may be of extreme benefit in diagnosis, that is, arteriovenous aneurysm of the lung, but to this date we have not had any experience in this category.

ANNOUNCEMENT

It is a pleasure to announce that arrangements have been completed which will make possible the conversion of the JOURNAL from a bimonthly publication to one which will appear monthly. This change will begin with the first number of Volume 19, January, 1950. The C. V. Mosby Company has generously granted to The American Association for Thoracic Surgery not only the privilege of having regularly in its official journal 108 pages in each monthly issue but also the opportunity to publish during the year 1950 approximately 50 more pages in each issue in order to clear up a large number of accumulated papers which have been held for long periods because of the previous restricted number of pages. It is expected, therefore, that from now on it will be possible to print accepted articles much more promptly than previously.

In order to make the change to a monthly publication it will be necessary to increase the subscription price in the United States from $10 to $15 per year. The Council of the Association has authorized this increase.

The enormous interest in thoracic surgery which now exists throughout the world and the great speed with which it is being developed amply justify the change in policy of the JOURNAL. I feel certain that those who are interested in this field of surgery will welcome it.

—The Editor

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1949-1950

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NOTICE

The thirtieth annual meeting of the American Association for Thoracic Surgery will be held April 15, 16, 17, and 18, 1950, in Denver, Colo. Headquarters will be at the Cosmopolitan Hotel.

Requests for hotel reservations should be mailed direct to Col. James H. Forsee, 519 17th Street, Denver, Colo.

Abstracts for presentation of papers for the 1950 meeting must be received for consideration by the Program Committee on or before Jan. 1, 1950. These abstracts should be submitted in *quadruplicate* and addressed to the Secretary of the Association, Dr. Brian Blades, George Washington University Hospital, 901 23rd Street, N. W., Washington 7, D. C. They should not be sent to the Editor. Abstracts received after Jan. 1, 1950, will not be considered.

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